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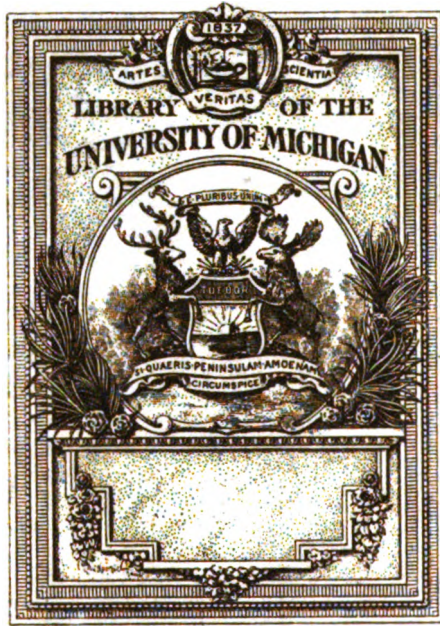
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FIG. 1.



Hermetically sealed tube of "606". (Natural size and color, showing dosage and control marks.)

FIG. 2.



Microscopical appearance of "606" after exposure to the air and its attendant moisture, with decomposition products especially seen on the right and at the edges of the undecomposed arsenobenzol. (Ehrlich insists on the importance of freshly preparing the salt just previous to the giving of an injection.)



# **INTERNATIONAL CLINICS**

## **A QUARTERLY**

OF  
ILLUSTRATED CLINICAL LECTURES AND  
ESPECIALLY PREPARED ORIGINAL ARTICLES  
ON  
TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-  
RICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS,  
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,  
OTOLOGY, RHINOLOGY, LARYNGOLOGY,  
HYGIENE, AND OTHER TOPICS OF INTEREST  
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION  
THROUGHOUT THE WORLD

EDITED BY

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 Microscopical appearance of "606" after exposure to the air and its attendant moisture, with decomposition products. (Fig. 2)....*Frontispiece.*

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# Diagnosis and Treatment

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## EHRLICH'S NEW PREPARATION, ARSENOBENZOL ("606"), IN THE TREATMENT OF SYPHILIS \*

BY HENRY W. CATTELL, A.M., M.D.

Editor of Lippincott's New Medical Dictionary; Fellow of the College of Physicians of Philadelphia, etc.

---

THE past six years have witnessed four distinct advances in our knowledge of syphilis. First came the epoch-making discovery by Schaudinn and Hoffmann of the *Treponema pallidum* or *Spirochaeta pallida*, the causative agent of this wide-spread affection. The recognition of this organism has been greatly facilitated from the use of the dark-stage illuminator and by the azure stain of Giemsa and by the silver nitrate method of Levaditi. Then the animal-inoculation of apes by Metchnikoff and of guinea-pigs by Uhlenhuth fulfilled another of Koch's postulates, while the Wassermann reaction made control tests possible in those affected with syphilis. And now come Ehrlich and Hata, of the Royal Institute for Experimental Therapeutics at Frankfort-on-the-Main, with a synthetical compound, the diamidodihydro-arsenobenzol dihydrochloride for the treatment of syphilis, which preparation is superior to atoxyl, arsacetin, and arsenophenylglycin ("418") as a spirilloicide.

The method employed by Wechselmann at the time of my visit to Berlin towards the end of July is reported in the July 28, 1910, issue of the *Deutsche medizinische Wochenschrift* and the Sept. 3, 1910, number of the *New York Medical Journal*. The contents of the hermetically sealed glass phial consisting of a previously weighed amount of the yellowish powder dioxydiamido-arsenobenzol (*Fron-*

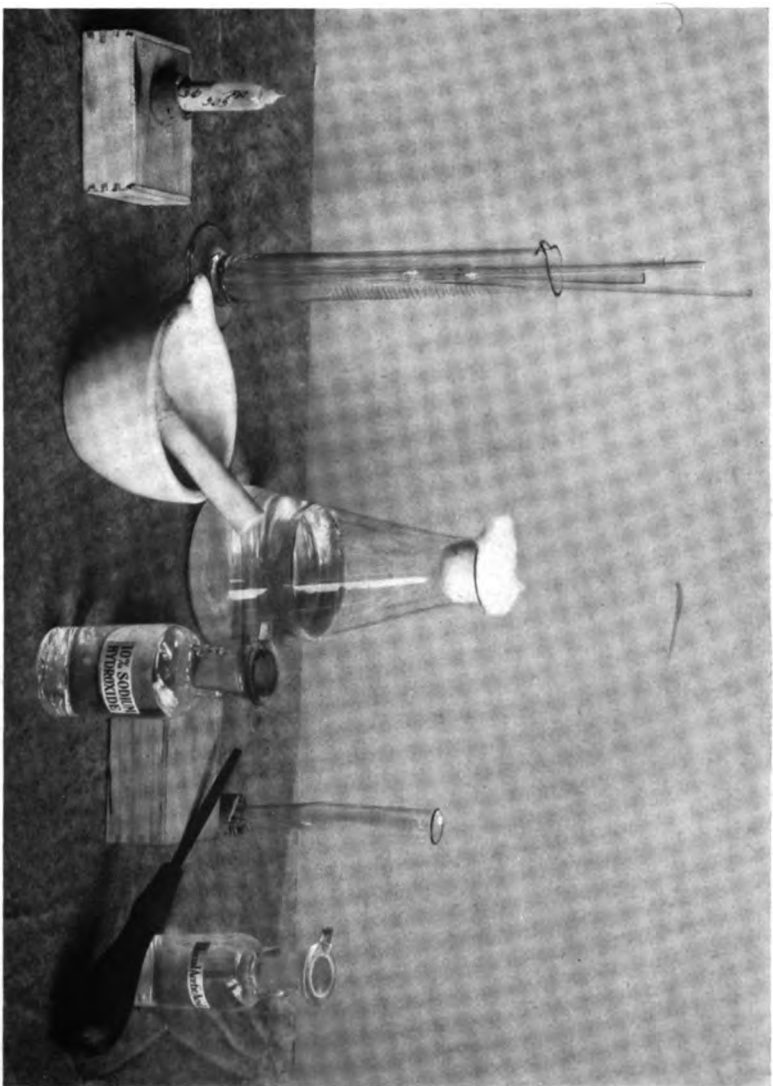
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\* A paper read before the Phila. County Medical Society, Sept. 28, 1910. (For literature, see Fordyce, N. Y. Med. Jour., Nov. 12, 1910.)



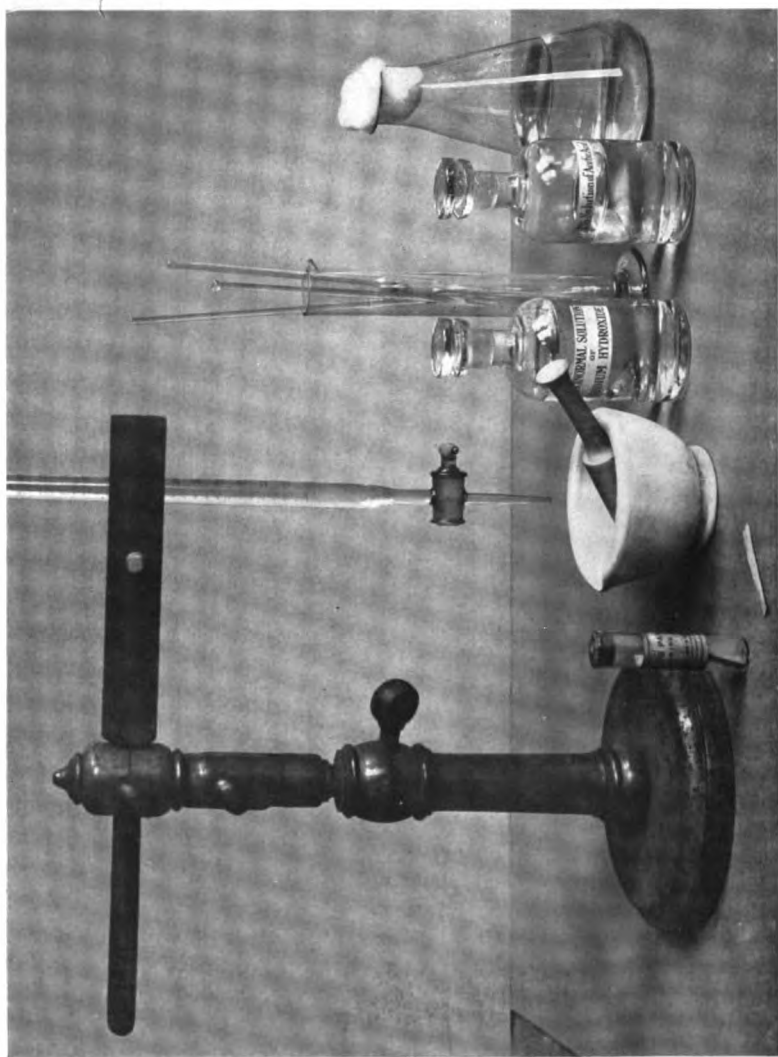
*tispiece*, Figs. 1 and 2), are placed in a mortar, and one cubic centimetre (half a drachm) of a sterile ten per cent. solution of sodium hydroxide added at a time until solution occurs. This is done because the salt is very prone to undergo decomposition on exposure to the air, and because the mono- or disodium salt is more suitable for the purpose of injection. Glacial acetic acid is next added drop by drop until no more of the yellowish precipitate is produced. About 15 c.c. (half an ounce) of water are then added, rubbed up in the mortar until evenly suspended, and the whole centrifugated and the supernatant liquid poured off (Fig. 3). The precipitate is then washed into a sterile dish and water or physiological salt solution added to make about 25 c.c. (6 fluidrachms). This suspension of "606" is now to be made neutral by the addition of either a one per cent. acetic acid solution, if the reaction as determined with litmus paper is alkaline, or else with a decinormal solution of sodium hydroxide, if it be acid (Fig. 4). A large sterile syringe with a good-sized needle is employed. The previous boiling of the needle in olive oil is recommended by McDonagh. The one to whom the injection is to be administered sits on a chair with his head resting on the folded arms supported by the back of the chair (Fig. 5), or the patient may lie flat on his stomach on a couch with his hands above the head so that the syringe will not be grasped during the injection in case there should be any pain. The region under the shoulder-blade is to be preferred for the place of injection, and after the skin has been cleansed for the purpose of the injection, the surface is painted with tincture of iodine and the suspension prepared as above described *slowly* injected in the same manner as is done with diphtheria antitoxin. The advantage of this region is that the injection there causes much less pain than in the gluteal region, and that it is easily accessible from a surgical standpoint in case of the development of arsenism. Intravenous injections act more quickly, and if employed the dilution should be at least up to 250 c.c. ( $\frac{1}{2}$  pint). Some prepare two solutions for this purpose. Up to half a gramme (8 grains) of "606" is dissolved in 3 c.c. (50 minims) of glycol, and 240 c.c. (8 fluidounces) of normal saline solution are added to 10.3 c.c. (167 minims) of  $n/5$  sodium hydroxide (0.8 per cent. solution). Each of these solutions is well shaken, and mixed immediately before

FIG. 3.



Tube of "006" and the apparatus and chemicals needed for the preparation of the first stage of the remedy previous to injection. (See Fig. 4.)

FIG. 4.



Apparatus and chemicals needed in the second stage of the preparation of "906," previous to injection in order to have a neutral or slightly acid medium for the insoluble arsenical salt. (See Fig. 3.)

use. A strongly alkaline solution, especially when injected into the gluteal region, is quite painful, and untoward effects of the methyl alcohol, formerly used to dissolve the salt in place of the sodium hydroxide, have been reported by numerous observers. Hot ethyl alcohol may be used, however, one-half cubic centimetre for each 0.1 gramme of the powder employed (5 minims per grain). Alt uses thirty or forty small glass beads in making the emulsion, as the "606" is apt to collect in lumps if some such method as agitating with the beads or rubbing up in a mortar and centrifugating is not employed.

Most careful consideration must be given to proper dosage, it being intended that, if possible, a single dose shall kill the protozoa without being toxic to the affected person, the so-called *therapia magna sterilisans*. At the start too small doses were administered, and I saw in Wechsellmann's wards, Cases 1, 7, and 18 (reported in the *Dermatologische Zeitschrift*, xvii, No. 7, 1910), which had returned for further treatment. From careful experiments performed on the lower animals by Alt it would seem, if the same proportion should hold in the case of human beings, that it would take 7.0 Gm. (110 grains) to be lethal to a man of average weight and in good condition. Small doses of 0.2 to 0.3 Gm. (3 to 5 grains) have now yielded to the larger ones of 0.5 to 0.6 Gm. (8 or 9 grains), and Treupel suggests that the dose in selected cases will be even as much as 0.9 Gm. (14 grains). When the smaller doses were administered the rash and other lesions seemed at times to be rendered even worse from the use of this remedy. The dose for babes with inherited syphilis was first 0.015 Gm. ( $\frac{1}{4}$  grain), which was found to be too small, and then increased to 0.025 Gm. (0.4 grain). Before the drug is administered a careful physical examination of the one to be treated should be made, the pulse and temperature recorded, the eyes examined, and the existence of any complications in the liver, kidneys, or other vital organs determined. I would advise a day's previous rest in bed, securing the usual movement of the bowels, proper food, etc., so generally employed before any operation. If possible an attempt should be made to demonstrate the presence of the *Treponema*, and the Wassermann reaction should be determined.

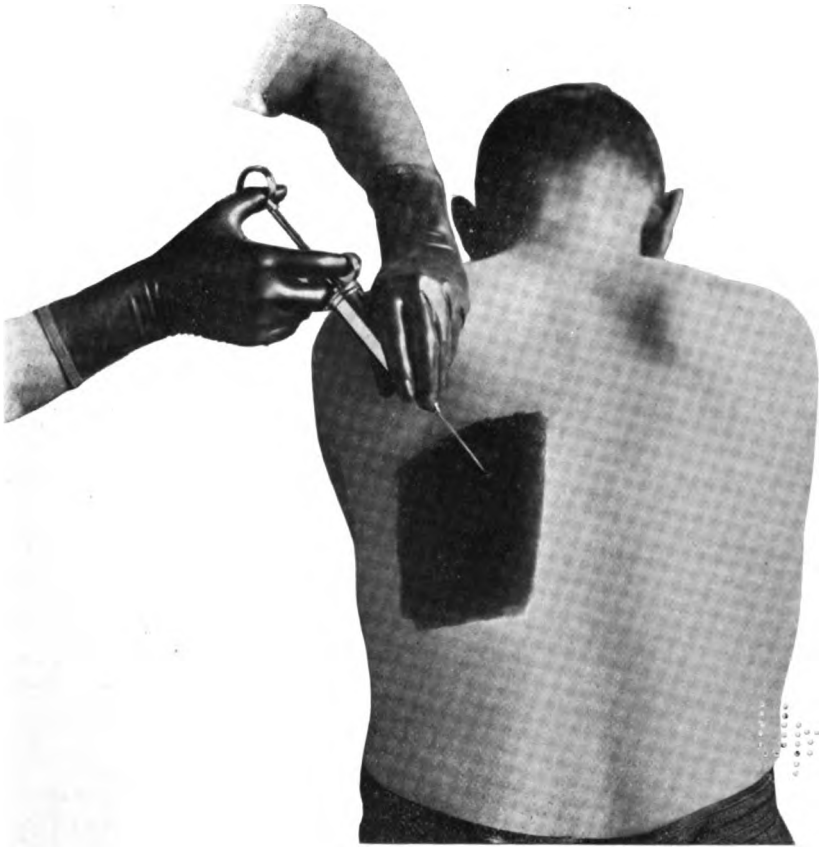
After 12 to 18 hours, if the dose is a proper one, the spirochaetes will begin to disappear, and in the course of several days they will no longer be capable of demonstration. The Wassermann reaction under similar conditions grows less and less marked, and in the course of several weeks becomes negative. In certain cases the organisms seem to be arsenic-fast and not capable of destruction. This occurs most frequently in those cases in which the first dose was too small or where other preparations of arsenic have been previously employed. J. E. R. McDonagh<sup>1</sup> determines whether the patient is likely to act badly or whether arsenic has been previously administered by tests similar to those used in the Calmette or Von Pirquet tests with tuberculin. To test for this oversensitiveness to arsenic a conjunctival (which I do not advise) or cutaneous reaction is undertaken with the drug in the strength intended to be used, or with a solution of arsacetin 0.3 gramme to 3 cubic centimetres of water (6 grains to the drachm). Should the reaction be positive, such patients should be regarded as unsuitable for the immediate treatment by "606."

And now as to the results thus far obtained. Ehrlich has reports on over 30,000 cases with some fifteen deaths, only a few of which he believes to have been caused by the arsenic itself. I saw in Wechsellmann's clinic lesions of various sorts, such as those of iritis, mucous patches and gummata, rapidly disappear under "606," and I have obtained similar favorable results in a few of my own cases so treated. As this method was employed for the first time less than one year ago, care must be taken about any unduly extravagant claims as to absolute cure. The experiments of Neisser on apes would show that an animal suffering with the secondary symptoms of syphilis might be completely cured of syphilis and again inoculated with the disease. Whether this will be true in the case of man will be quickly determined by observation of the Berlin prostitutes. This large class of women, which is kept more or less completely under police surveillance and subject to examination by physicians, is flocking to the hospitals for treatment, and if a cure is actually effected it will not be long before through their mode of life a second infection will take place and thus firmly establish the fact of the superiority of "606"

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<sup>1</sup> *Lancet*, September 3, 1910, p. 711.

FIG. 5.



Photographic illustration of method of giving an injection of "606," used in Wechselmann's clinic in the Rudolph Virchow Hospital at Berlin. The patient is sitting astride of a chair, with the head supported by his arms resting on the back of the chair. The previously cleansed skin has been painted with iodine; and the needle introduced, previous to bringing pressure on the piston of the syringe in order to inject the specially and recently prepared "606."

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over mercurials. Should such be the case it will be interesting to study the effect on the statistics of prostitution offered by the removal of the deterring influence of fear of contracting this disease. The effect on future generations will also be interesting. The drug administered to the mother does not find its way into the blood of the child, but protective principles are formed which do affect favorably the course of the disease in a nursing infant.

While attending Professor Orth's pathological demonstrations at Berlin this summer, we were shown the specimens from a patient who had died on the tenth day after an injection of ".606." The man was a tabetic, and showed marked arteriosclerotic changes in the arteries, with an ossifying pachymeningitis of the spinal cord, which plainly demonstrated with the naked eye degeneration of the posterior columns. The arsenobenzol had been injected into the buttocks, and there was an infiltrated necrotic area at the point of injection which was sterile from a bacteriological stand-point, there being no suppuration, but a similar condition of the tissues such as is seen after the injection of calomel or other insoluble preparations of mercury. This patient's physical condition did not, however, warrant the administering of the remedy.

#### CONCLUSIONS

1. From a study of the literature and from a personal examination of over a hundred patients in Berlin and Philadelphia, treated for syphilis with injections of arsenobenzol, I believe the advantages of this mode of treatment, in properly selected cases, outweigh the older methods of using mercury internally, or by inunctions, fumigations, or injections.

2. The attendant dangers are no greater than in the hypodermic use of the insoluble salts of mercury, while the fact that the therapeutic effects of a single treatment will last for from three months to a year, with the possibility of a complete cure, mark the nearest approach yet reached to the ideal remedy that will be parasitotropic, that is, kill *Protozoa* within the body without being organotropic, that is, injuring the body tissues themselves.

3. The discovery of this remedy demonstrates what may be obtained by painstaking research carried out under favorable conditions, and with the use of animal experimentation.



**TRUE SYPHILITIC IRITIS (IRITIS PAPULOSA)  
TREATED WITH AN INJECTION  
OF EHRLICH'S "606" \***

**BY G. E. DE SCHWEINITZ, M.D., AND E. A. SHUMWAY, M.D.**

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A COLORED man, aged 19, was admitted to the Philadelphia General Hospital, September 17, 1910, with iritis of the left eye. His family and personal history are unimportant and need not be detailed. He denied all illness and stated that he had always been a perfectly healthy person. In August, 1910, he developed three ulcers on the corona, and consulted a physician, who told him that the lesions were specific and ordered for him a lotion but gave no constitutional treatment. The Resident Physician who took the history was unable to elicit from the patient any information in regard to secondary lesions, that is to say, he gave no account of skin eruption, falling of the hair, or any similar symptoms.

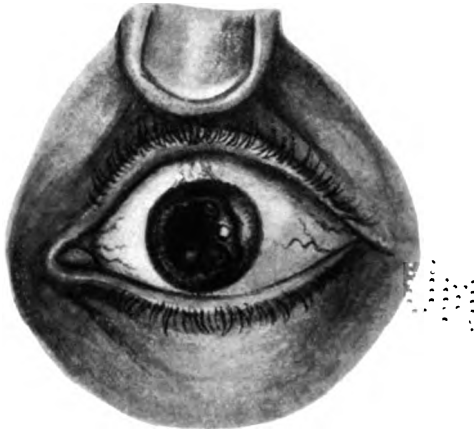
Three days after his admission to the Philadelphia General Hospital, namely, on September 20, inunctions of unguentum hydrargyri were ordered and six of these inunctions were given, and atropine was instilled into the eye. When Dr. de Schweinitz took charge of the wards on the first of October, the lesions depicted in the accompanying illustration (Fig. 1) were present, namely: A broad synechial attachment up and out, at the margin of which a small, yellowish-white nodule was apparent, which was the external end of an exudation which existed between the attached margin of the iris and its ciliary border. The ciliary border was swollen and discolored, and terminated in the groove which is evident in the illustration. Down and slightly in, there was a patch of lymph upon the capsule of the lens, marking, in all probability, the position of a synechia which had been broken loose by the atropine. The right eye was unaffected and healthy.

Protiodide of mercury was ordered, but fortunately, through a

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\* Case reported to the Ophthalmic Section of the College of Physicians, October 20, 1910.

FIG. 1.



Syphilitic iritis (*iritis papulosa*) before treatment,  
with the '606'.

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misunderstanding, was not given, so that the patient had no constitutional treatment except the six inunctions previously referred to.<sup>1</sup> On October 5 Dr. Laird made a Wassermann test, finding it actively positive, as follows:

REPORT OF WASSERMANN REACTION ON L. F., EYE WARDS, PHILADELPHIA HOSPITAL

October 5, 1910, positive.

October 7, 1910, quantitative.

	COMPLIMENT	HÆMOLYSIS	
Sp. antigen, 0.2 Pat. serum, 0.1	0.05 c.c. ....	0	Hæm. ambo., 1.0 Sh. blood, 1.0
	0.06 c.c. ....	0	
	0.07 c.c. ....	very slight	
	0.08 c.c. ....	very slight	
	0.09 c.c. ....	slight	
	0.10 c.c. ....	slight	
	0.12 c.c. ....	partial	
	0.15 c.c. ....	complete	
	0.20 c.c. ....	complete	

0.05 c.c. compliment = 1 unit of syphilitic amboceptor.

Result: Positive.....1½ units.

Through the courtesy of Dr. Coplin, one of us (Dr. de Schweinitz) obtained the promise of  $\frac{6}{10}$  of a gramme of Ehrlich's "606," and treatment of all kinds was stopped until October 10, on which day Dr. Funk, Dr. Coplin's assistant, very kindly came to the hospital and gave beneath the right scapula a single injection of "606," about four o'clock in the afternoon. Until midnight of the same day the patient complained of much pain, and vomited two or three times. After that the pain ceased, and since that period the patient has suffered neither with his eye nor in any other way.

Within forty-eight hours the eye had grown whiter and the thickened iris less conspicuous, and the whitish area had almost disappeared. It is, however, proper to state that in the period between the third of October and the tenth of October, on which day the Ehrlich injection was given, although the patient was having no treatment whatsoever except atropine locally, there had been some

<sup>1</sup> Since this was written we have ascertained that he also took a small quantity of iodide of potassium; according to the Resident not more than 20 grains.

improvement in the eye, and the lesions on the tenth were somewhat less conspicuous than those which are depicted in Miss Washington's drawing made on the third. On the other hand, the improvement after the tenth until the present time has been so rapid that it is difficult to believe that it could have been due to the six inunctions of unguentum hydrargyri which had been given from the twentieth to the twenty-sixth of September, no other treatment having been used either before that time or after that time except atropine locally, and the atropine was stopped on the tenth of October. The vision at the present time is O. D. 20/20, O. S. 20/40. Unfortunately, there is no record of vision taken on the day of the patient's admission, but we suspect that it was not greatly different from that which is now recorded, because the major portion of the pupil had not been blocked with lymph, and although there were some precipitates on the posterior layer of the cornea, they were not very thick.

Nine days after the injection a Wassermann test was positive ( $\frac{3}{4}$  units). The patient left October 20, 1910, and has not been seen since.

# THE UNITED STATES PHARMACOPŒIA (NINTH REVISION)

BY JOSEPH P. REMINGTON, Ph.M.

Chairman of the Committee of Revision of the U. S. Pharmacopœia; Professor of Theory and Practice of Pharmacy, and Director of the Pharmaceutical Laboratory in the Philadelphia College of Pharmacy

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THE Decennial Convention for revising the Pharmacopœia has come and gone. Notwithstanding the turmoil wrought by a few agitators which stirred the surface within the last two years, it is surprising to note how little the depths of the waters were moved.

The rank and file of the medical profession were not much in evidence at Washington. The professors were there and some of the specialists, but it might as well be admitted now as at any other time that the attempt on the part of a few persistent touts, who predicted all sorts of calamities if the Convention did not do as they ordered, amounted to nothing, and the fact remains as it has for thirty years that the preponderating influence in the work of revision must, necessarily, be pharmaceutical and chemical.

There was no attempt made to curtail medical influence in any way, speakers were treated with courtesy by everyone, but the influence of the clinician and the practising physician was absent; much as we regret this, it must be admitted that the Pharmacopœia has become, mainly, a book of standards and as such it must wield, in the future, a vast influence in improving the quality and establishing the uniformity of medicines supplied to the medical profession.

The election of Dr. Harvey W. Wiley to the presidency was a logical move, for it directly connects the most prominent leader in the Pure Food and Drug reform with the book which is used as the legal standard, and the basis for prosecutions in Nation and State. The presiding officer, Dr. Otto A. Wall, of St. Louis, was an able parliamentarian, firm, impartial and prompt and although a larger amount of business was conducted than at any previous

convention, only about one-half the usual time was consumed. Nearly all of the members of the previous Committee of Revision were re-elected by large majorities, but a change was made in enlarging the general committee to fifty and from these, selecting fifteen to constitute an executive committee, which is delegated to do the actual work of revision; this plan should produce better results and save time. The subcommittees have been elected and the real work of revision is now under way.

## FURTHER CONTRIBUTIONS ON THE TREATMENT OF LEUKÆMIA BY THE RÖNTGEN RAYS \*

BY HENRY K. PANCOAST, M.D.

PHILADELPHIA

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WHENEVER one's attention is called to the subject of Röntgen ray treatment in leukæmia there are two thoughts that are very apt to be aroused in connection therewith. In the first place, one is reminded of the profound and striking effects of radiation upon the enlargements and the leucocytosis, and often the apparent production of a complete disappearance of all manifestations of the disease, all of which for a short time lead to the belief that a permanent and specific curative agent had at last been found for this incurable malady. As a second thought, however, one is apt to recall also the fact that X-ray treatment has invariably failed to alter the ultimate prognosis in the disease, although radiation has proved to be the most satisfactory and successful method of treatment yet devised. If one is content to carry his thoughts no further than this, he has not only established his own conclusions regarding the exact limitations of X-ray therapy upon such a basis, but has based these conclusions upon the past results derived through methods that originated in mere experiment, and which, from the stand-point of the most generally accepted ideas concerning the pathology of the disease, have not been founded upon the most rational principles. To some who stop to consider these results of the past from such a stand-point, together with the remarkable effects of radiation upon the characteristic manifestations of the disease that have resulted from an abnormal cell proliferation, it must certainly seem reasonable to expect that more good might be accomplished through the use of a more scientific method of application of the treatment.

A method of applying radiation that seemed to be based upon

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\* This article embodies, with certain additions, the essential facts presented in a paper read before the recent annual Convention of the American Röntgen Ray Society, at Detroit, September, 1910.



more rational and certainly upon more scientific principles than the manner in which X-ray treatment had previously been employed, and which seemed, therefore, to promise better results than had hitherto been attained, was first suggested to the author by Dr. Stengel, nearly four years ago, and embodied as its essential principle the fact that the exposure of the bone-marrow should be regarded as an object of far greater importance in the treatment than the direct radiation of the spleen and other secondary enlargements. The technic that was developed for the embodiment of this suggestion has been employed exclusively in connection with all of the twenty cases of splenomyelogenous and lymphatic leukaemia since treated.

The essential object of this article is to present a few personal observations that have been made from time to time in connection with the treatment of these cases, and to substantiate our claims for the superiority of this method over that previously used by citing a few facts based both upon these observations and the general results so far realized. It is unnecessary to reiterate in detail the numerous arguments, based mainly upon the pathological features of the disease and the physiological action of radiation upon normal and abnormal lymphoid structures that were advanced when this method was first advocated in the preliminary report presented conjointly with Dr. Stengel three years ago.<sup>1</sup> The reasons for the adoption of the above suggestion, and the continued use of the technic which was developed for the purpose of carrying it out, may be briefly enumerated as follows:

In the first place, an acceptance of the views prevailing at the present time concerning the pathology of leukaemia implies a conception of its nature as that of a wide-spread malignant disease, with the primary foci of cell proliferation in the bone-marrow, and the splenic and lymphatic enlargements as representing secondary metastatic foci of proliferating cells originally derived from the bone-marrow, and subsequently augmented by further deposits brought from the same source by the blood stream. On the basis of such a conception, it would seem reasonable to suppose that, in order to attempt a permanent cure of the disease, the primary

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<sup>1</sup> See Stengel and Pancoast, *Trans. Am. Röntgen Ray Soc.*, 1907, and *Jour. Am. Med. Assoc.*, April 25, 1908; and Pancoast, *Ther. Gaz.*, Aug. 15, 1908.

object of the treatment should be the inhibition of further cell proliferation in the bone-marrow, which would seemingly be accomplished more directly by the exposure of the bones themselves than by limiting the treatment almost entirely to the secondary enlargements, where, though it produces a somewhat similar inhibitory effect on the primary foci, it does so in an indirect manner, through the production and subsequent action of leucolytic substances.

Secondly, the direct exposure of the bone-marrow would seem likely to induce a more powerful inhibitory effect than these leucolytic substances, and our observations would lead us to believe that not only is this true, but that the effect is more lasting as well.

Thirdly, as this direct inhibitory effect of the exposure of the bones alone seems to prevent, or at least limit, further metastasis, we are enabled to postpone the direct applications to the spleen and other enlargements until a later period, when the exposure of these enormous masses of extremely susceptible cells to direct radiation is comparatively safe, owing to the improved general condition, the decreased leucocytosis, and the reduction in size of the enlargements which so often occurs before the time for their direct exposure is reached. In this way, the dangerous, and frequently fatal, toxæmia which is so apt to follow the early radiation of the spleen and other enlargements is entirely avoided.

Fourthly, owing to the combined effects of the direct exposure of these structures, of a rapid reduction in their size, and the indirect inhibition of cell proliferation by the bone-marrow, an apparent disappearance of the manifestations of the disease, or the so-called "symptomatic cure," is produced too soon, whenever such a result is obtained, for any permanent influence over the primary foci to have been established. On the other hand, it takes very much longer to bring about apparently the same symptomatic result by the bone-marrow treatment, and during all of this additional time a constant attack is kept up upon the primary foci, thereby insuring a far more effectual control over the disease.

Fifthly, the more extensive distribution of the applications over practically the entire surface of the body reduces the risk of a "burn," or X-ray ulcer, to a minimum. The skin should be carefully watched, however, and especially where the exposures of contiguous areas may overlap.

Sixthly, the results achieved by this method, though by no means satisfying as yet, have, on the whole, proved far superior to those generally effected by the older method.

*Technic.*—With the omission of all details that have been previously presented in the preliminary report, the technic of the treatment, as it was first advocated, and has since been modified through further observations and experience, is essentially as follows:

1. The applications are made primarily over the bones of practically the entire skeleton.

2. Each area is exposed regularly and systematically, and it has seemed advisable, from numerous observations made in connection with the effect of the applications upon different manifestations of the disease in addition to the reasons originally advanced, to expose each area at least three successive times during each series of applications over the body.

3. Exactness in dosage measurement and administration is essential, especially during the early period of the treatment, when toxæmia is readily induced. While this effect should always be avoided, it is advisable, on the other hand, to administer the maximum dosage possible without the production of the least toxic manifestations.

4. Continued experience leads us to believe that frequency is one of the most important factors in successful treatment. Except in instances of severe toxæmia, daily applications cannot be too strongly advocated. We have to deal with a wide-spread disease of a malignant character, in which the structures involved are in a constant state of most active cell proliferation, and the inhibitory influence of safe radiation upon the abnormal cells and the abnormal processes involved is not of very long duration. As it is necessary to treat only comparatively small areas of the body at a time, we must depend largely upon the cumulative action of radiation for a continuance of the effect between the times of two sets of exposures to any one part while the rest of the body is being covered, and if the applications be made less frequent than almost daily, this intervening period will be too prolonged for the effect of the previous set of applications to any part to continue, hence the treatment will not be able to exert much influence over the disease.

5. Toxic reactions are always harmful, and have frequently proved fatal, and therefore should always be avoided. Toxæmia is an indication for a temporary lessening of dosage, rarely for a lessened frequency of the applications, and never for a cessation of treatment unless it is to be discontinued permanently. The addition of a very few milliampère-minutes to a safe dosage may be sufficient to induce a toxæmia, and even an albuminuria, as has been noted in at least one instance.

6. The direct exposure of the spleen or those regions containing other lymphatic enlargements should be carefully avoided during the early period of the treatment, for the three reasons already given: first, because the exposure of the bone-marrow is of primary importance; secondly, because of the dangers resulting from the toxæmia so apt to be induced; and thirdly, because the rapid decrease in leucocytosis and the size of the enlargements, and the rapid disappearance of these and other manifestations of the disease are apt to be misleading in inducing a cessation of treatment at entirely too early a period. The secondary enlargements should be exposed at some time, of course, but this should be left until a safe period in the treatment, when some diminution in their size has taken place, as is very apt to be the case, and the leucocytosis has materially diminished, on the one hand, thus rendering toxic reactions less likely to be induced; while on the other hand, a decided improvement in the general condition permits the increased liberation of waste tissue products to be better borne.

7. Prolonged periods of rest from treatment are to be discouraged on the part of the patient and avoided by the röntgenologist. There should be no cessation in the applications until it is time to stop, or there is no likelihood of any further good being accomplished.

8. Our own experience leads us to believe that the duration of the treatment is one of the most important factors bearing upon the ultimate prognosis in favorable cases. All of us must realize from past experience that in hardly a single instance where a case has responded favorably to the treatment has a normal leucocyte count, a disappearance of splenic and glandular enlargements, and a return to an apparently normal general condition of health,—or, in other words, a symptomatic cure,—failed to be followed sooner

or later by a relapse, and ultimately by death. Why, then, should the treatment be stopped at such a period, as has invariably been the case, to say nothing of hastening the attainment of such misleading results by following the older method of treatment? In the treatment of any malignant growth, recurrence is an inevitable sequence unless every vestige of the disease is eradicated. If, then, we are to regard leukæmia as of the nature of a malignant condition, and in no other one does radiation exert such a powerful effect upon the cellular elements, it should be treated in the same manner as any other condition of a malignant type, which means, first, that the attack should be continued until not the slightest vestige remains, and secondly, that it should be resumed on the appearance of the first warning of an approaching relapse. One vestige that frequently remains after the attainment of the so-called symptomatic cure is a more or less abnormal differential count, and under such circumstances the applications should be continued vigorously until normal ratios are established, as without these permanent cures certainly need never be expected. Furthermore, by carefully watching the differential count subsequently, the first warning of an approaching relapse may be readily recognized in a tendency toward a reversion to abnormal ratios. Experience has proved that one or two series of applications over the entire body at such a time, before there is even a rise in the leucocyte count, will bring about a return to practically normal ratios, and ward off the approaching relapse, which is certainly more readily and more quickly accomplished than is the successful treatment of a definite relapse. It is our belief that unless the final treatment of favorable cases be so governed by the differential count, no permanent good need ever be expected from X-ray therapy in leukæmia. Unfortunately, we are not always able to carry the treatment to such a favorable issue, and furthermore, whenever such a point is reached, so is the limit of our present knowledge as to what to do further if this prove insufficient for the attainment of our highest aims. It may be well to call attention at this time to the fact that it occasionally happens that the leucocyte count drops to normal or even subnormal figures while some manifestations of the disease, such as a slight enlargement of the spleen or the glands, still persist, but one should not hesitate to continue the treatment further on

this account as long as any indication for its continuance exists, especially abnormal ratios in the differential count.

9. The administration of arsenic in conjunction with X-ray treatment is a matter that deserves more careful attention than it has ordinarily received, for undoubtedly there is a decided danger as well as a distinct value in the use of this drug under such circumstances. As a rule, there can be no objections to its administration in small or so-called tonic doses during the later periods of the X-ray treatment, provided it exhibits no tendency to derange the digestive functions in any way. The administration of any form of arsenic, on the other hand, in the large doses such as are ordinarily regarded as necessary to produce the proper therapeutic action in this particular disease, cannot be regarded as a wise or even a safe routine procedure, especially during the early period of X-ray treatment. In fact, large doses of this drug should never be administered in conjunction with radiation without due consideration of the similar destructive actions of these two agents, and the double destructive effect that they are likely to induce. Arsenic in large doses is capable of destroying leukæmic cells in much the same manner as is direct radiation, and of indirectly inducing a similar inhibitory effect through the resulting production and liberation of leucolytic substances. Moreover, the toxins so produced, added to those derived from the radiation, may be sufficient to induce a dangerous toxæmia. Furthermore, this similar action of the arsenic may not only interfere temporarily with the proper adjustment of X-ray dosage, but may also be responsible for a mismanagement of the treatment throughout its whole course, first, by lowering the leucocytosis too rapidly and thereby rendering any exact determination of the effect of the radiation difficult or impossible; and secondly, by promoting too rapid a disappearance of the manifestations of the disease it may lead to a cessation of the treatment at too early a period. Further than this, the disturbances in the digestive functions that are likely to result from the administration of large doses of arsenic may tend to interfere seriously with the proper elimination of the waste tissue products. The one great value of arsenic in conjunction with X-ray treatment, aside from that as a tonic, is in connection with cases that are favorably influenced by radiation up to a certain

point, and then remain practically stationary, X-ray applications seeming to have no further influence over the disease. In such instances, the administration of arsenic in moderate doses will frequently supply the deficiency and materially assist the radiation in bringing about further improvement. Also, in connection with cases in which X-ray treatment, for certain reasons having no bearing on the latter, has to be discontinued at too early a period, arsenic is the only means of delaying the relapse that is sure to follow soon, and its use is always advisable from the stand-point of prolonging the life of the individual as long as possible. Such patients should be induced to have X-ray applications whenever possible, however, even if it be but one or two a month.

10. X-ray treatment is practically contraindicated in the acute forms of the disease; and in the late stages of chronic cases, during either the primary attack or a relapse, when an extreme degree of toxæmia exists. The additional waste products resulting from radiation under such circumstances are likely to cause an overwhelming toxæmia in any such case, and to hasten death thereby.

*Prognosis.*—There can be no doubt that radiation, when carefully and judiciously applied, is the most efficient method of treatment so far devised for dealing with this disease, and yet all of us are well aware of the short-comings of X-ray therapy in the past. It is earnestly to be hoped, however, that a wider experience in the use of this agent and a better understanding of the true nature of the condition as a basis for further improvements in the methods of applying the treatment will render a permanent control over the disease possible in the near future, unless, of course, a more efficient therapeutic agent is discovered in the meantime. A permanent cure is the result most desired, of course, but it is difficult, or impossible perhaps, to define accurately the exact meaning of such a term used in connection with this particular disease, for the reason that we are ignorant of the exact identity of the primary etiological factor. So far as we can reasonably assume, a permanent cure implies, in addition to a complete symptomatic cure, a permanent destruction of or control over the active cause of the disease. We can rationally hypothecate as to the cause in this disease, and possibly in polycythæmia also, so far as to comprehend it either as a loss or lack of some controlling influence which normally regulates cell proliferation, and at such a rate as to maintain

the normal ratios that should exist between the various blood elements; or, on the other hand, as an abnormally existing or active factor which directly or indirectly inhibits or otherwise disturbs this controlling influence. Upon the basis of such hypotheses, radiation, to be ultimately successful, must exert such an effect as either to restore what is lost or lacking, on the one hand, or, on the other, to destroy or render inactive some definite, but so far unrecognized, etiological factor. In the absence of any more exact conception of the cause of the disease, our only certain means at the present time of estimating the chances of a satisfactory result having been attained is our success toward the complete elimination of every apparent manifestation of the disease.

All that can be conscientiously claimed as having been definitely accomplished so far by X-ray treatment, aside from the temporary symptomatic cures that have been realized in a reasonably large percentage of cases, may be regarded as an inhibitory influence whereby life has been prolonged for variable but definite periods of comparative comfort. It is to be hoped that our failure to obtain permanent cures has been due to failure either to apply the treatment properly or to continue it long enough to eradicate the disease, as in the case of recurring malignant growths. Otherwise, radiation cannot be regarded as any more than a palliative measure. If it must be regarded merely as a palliative measure, our experience has led us to believe that the method of treatment herein and previously advocated offers a longer expectation of life in favorable cases than did the one we have seen fit to discard; that it is attended by much less risk to the patient from either toxæmia or "burns"; and that the general condition improves earlier, usually beginning to do so from the start, reaches a higher standard, and is maintained for a longer period of time. In favorable cases, the patients are able to resume active work during the progress of the treatment. The avoidance of toxæmia in the early period of treatment is no doubt an important factor in this connection. The leucocytosis diminishes and the enlargements subside much less rapidly, but this is regarded as an advantage rather than otherwise, as has been explained. The treatment by this method requires at least twice the length of time, or at least double the number of applications, in order to obtain apparently like symptomatic results, but this also has its distinct advantages.



# THE METHODS OF EXAMINING THE BLOOD OF GREATEST IMPORTANCE FOR THE GEN- ERAL PRACTITIONER

BY LEWELLYS F. BARKER, M.D.

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Johns Hopkins Hospital, Baltimore

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KNOWLEDGE concerning the blood has been making strides with great rapidity during the past few years; so many new methods of examining the blood have been introduced that the general practitioner, unless in close touch with special workers, is likely to be confused and to have difficulty in sifting out what is important for everyday use from what is ephemeral or what is suitable only for investigative work. It has seemed to me, therefore, that a brief article drawing attention to the methods of most importance to the general practitioner along with some comments upon the results which these methods yield, might be of service to those who are not actively engaged in hospital work. Of course opinions differ, even among hospital workers, as to the relative values of different methods, and the list of the most important methods prepared by one clinician may include certain methods which would be rejected by another, or exclude some methods which another would recommend. The list which I shall give is based upon our clinical experience in the Johns Hopkins Hospital, and includes only those methods which at the present time seem to me, personally, to be of sufficient importance to claim the attention of the physician in active practice. It is quite possible that in two or three years other methods, which as yet do not seem worthy to be included in this list, may have proved themselves of sufficient practical value to be added, or on the other hand, our clinical view-point may so change that some methods upon which we now lay emphasis may later cease to have the value at present attached to them.

The methods most important for the practitioner at present can, in my opinion, be summed up under the five following headings:

(i) the microscopical examination of fresh, unstained blood; (ii) the counting of the red and white blood-corpuscles; (iii) the estimation of the amount of hæmoglobin present; (iv) the study of the formed elements of the blood in dried and stained preparations; and (v) certain bacteriological and biological examinations of the blood, including (1) the making of blood cultures; (2) the testing for the presence of agglutinins in the serum (the Widal reaction); and (3) the testing for the presence of antibodies to the syphilitic antigen by the complement-fixation method (the Wassermann reaction).

A second group of methods less often applicable by the general practitioner, but still of sufficient importance to merit his attention, includes (*A*) the determination of the coagulation time of the blood; (*B*) the determination of the viscosity of the blood; (*C*) the determination of the resistance of red blood-corpuscles to anisotonic solutions; and (*D*) the catalase reactions.

While the above lists may seem tolerably comprehensive to practitioners who received their training before the establishment of the modern clinical laboratory, the number of methods included is in reality very small when one considers the whole series of methods which may for one purpose or another be resorted to in the clinical examination of the blood.

It may be interesting to append a list of some of the methods which I have excluded, not because they are of no importance,—many of them are of value in scientific clinical work,—but because one rarely needs to resort to any one of them for practical clinical diagnosis. Among the methods which the general practitioner in active practice may, as yet, very well dispense with may be mentioned; (1) the estimation of the total amount of blood in the body; (2) the determination of the specific gravity of the blood; (3) the determination of its alkalescence; (4) the determination of the osmotic pressure or molecular concentration of the blood (cryoscopy) and of the electrical conductivity of the serum; (5) the determination of the dry residue; (6) the determination of the protein content; (7) the quantitative determination of the sugar in the blood; (8) the quantitative determination of fat; (9) the quantitative determination of uric acid in the blood; (10) the estimation of the volume per cent. of blood-plasma and of blood-cor-

puscles; (11) the iodine reaction of the leucocytes; (12) various fermentation reactions in the blood; and (13) the estimation of the mineral salts.

It seems to me much better for the busy practitioner to familiarize himself with a few of the extremely important methods, becoming a thorough master of these, rather than to attempt a larger series and fail of precision in any of them. One method properly known and practised will give better results for diagnosis and prognosis than half-knowledge and half-skill in a hundred methods.

In obtaining blood for examination one is guided by the amount required for the method employed. When only small amounts are desired (blood counts, hæmoglobin estimations, fresh blood slides, smear preparations) sufficient can be obtained by puncture of the finger-tip or the lobule of the ear with a needle or the point of a small scalpel after thorough cleansing with ether and drying. The blood should, however, exude without strong pressure; mistakes are often made when strong pressure is used to force out the drop, since this pressure squeezes out lymph, which dilutes the blood. For blood cultures and for the Wassermann reaction it is desirable to have a larger quantity of blood and this is best obtained by inserting the needle of a sterile syringe into the median vein at the bend of the elbow, after surgical sterilization and the application of a tourniquet (causing venous compression) upon the upper arm. One can easily withdraw 50 to 150 c.c. (2 to 5 fluidounces) of blood in this way, an amount which suffices for all ordinary bacteriological and serological examinations. Wright's collecting tubes may be employed in the securing of the blood for the Wassermann test.

#### I. MICROSCOPIC EXAMINATION OF FRESH, UNSTAINED BLOOD

Of all the methods employed this is perhaps the one which is capable of giving most information to the busy practitioner, provided he knows how to avail himself of the results which the method can yield. The skilled observer, working with this method, can quickly gain an idea of the forms of red and white blood-corpuscles present, of the relative number of white and red cells and blood-platelets, and of the presence or absence of malarial parasites and of pigment. A few small points in the technic are of importance. The slides and covers used should be thoroughly cleaned with alcohol

and ether and the covers should be thin enough for use not only with lenses magnifying 300 times, but also with oil-immersion lenses. The drop taken up by the cover-glass should be so small that when it is placed upon the slide and runs out to a thin layer it does not extend as far as the margins of the cover-glass. It is desirable to have a preparation in which the red corpuscles can be seen lying singly, flat, beside one another, though at the periphery of the specimen rouleaux may be visible in places. In a properly prepared specimen of normal blood only one or two leucocytes will be visible in a single field of the microscope, and if one sees more than this, say five, twelve or twenty, one can be sure that a hyperleucocytosis (pyogenic infection) or a leukæmia of some form exists, and the necessity of an accurate count of the white corpuscles will be obvious. With practice it is possible to recognize the principal forms of white corpuscles in the fresh, unstained specimen and to decide whether the majority of them are of myeloid or of lymphadenoid origin, though dried and stained specimens are necessary for accurate discrimination among all the varieties of white cells.

The *form* and *color* of the red blood-corpuscles is best seen in this fresh blood slide. Distortion of the red cells (*poikilocytosis*) and inequalities of size (*anisocytosis*) due to the presence of abnormally large cells (*macrocytes*) and abnormally small cells (*microcytes*) can be made out at a glance. A little experience, too, informs one as to the relative individual corpuscular holding in hæmoglobin. If the red cells are very pale, as in the secondary anæmias and in chlorosis, one can be sure that the *color-index* (*vide infra*) is less than 1; on the other hand, if the corpuscles are of a deeper yellow than normal, and especially if many macrocytes be present (as in pernicious anæmia) it is probable that the color-index is greater than 1.

In malarial districts the examination of the fresh blood slide is a quick and satisfactory method for the diagnosis of the presence or absence of a *malarial invasion*. Since general practitioners have been better trained in the technic of this method, the reported morbidity from malaria has greatly decreased and that of typhoid fever has considerably increased. Even in malarial districts much less quinine is used than formerly in continued fevers. If a malarial infection exists it is, as a rule, easy to make the diagnosis from

the fresh blood preparation alone; its exact nature, whether tertian, quartan, or æstivo-autumnal can be decided upon, and, from the character of the parasites present and the stage of their development, very accurate prophesies as to the time of the next paroxysm and as to the intervals between paroxysms if the case remain untreated can be made. The practitioner soon learns to recognize the *tertian parasite* with its fine, yellowish-brown pigment; especially helpful for identification is the pallor of the red corpuscle in which it is contained and the tendency of infected corpuscles to have a larger diameter than their fellows. In the *æstivo-autumnal infections*, on the other hand, the observation of a crescent or of an ovoid form immediately establishes the diagnosis, and even when the hyaline, amœboid types alone are seen inside the corpuscles the deep color of the infected red cells and their tendency to contract rather than to expand, distinguishes this type of infection from the tertian type. *Quartan infections* are much rarer in this country, but when met with the parasites are recognizable by the very dark, coarse pigment granules and small number of spores in the segmenting forms. If the parasites are few in number and there is any doubt as to their nature, dried and stained specimens should be studied (Giemsa stain) with an oil immersion lens and a mechanical stage.

## II. COUNTING THE RED AND WHITE CORPUSCLES

For this purpose the practitioner must have the *Thoma-Zeiss blood-counter* and it is desirable in addition, for leucocyte counts, to have a slide with the squares marked off in the way suggested by Türck, though this is not absolutely necessary. Formerly the red and white cells were counted in the same preparation, but much more accurate results are obtained when separate dilutions are made for the two counts, that for the red count being a dilution of 1-100 and that for the white count being a dilution of 1-10 or 20. The best *diluting fluid for the red corpuscles* is that having the following composition: bichloride of mercury 0.5, sodium sulphate 5.0, sodium chloride 1.0, and distilled water 200.0. As a *diluting fluid for the white corpuscles* one uses something which will dissolve the red cells, preferably acetic acid. A satisfactory formula for such a diluting fluid is that of Türck, who uses glacial acetic

acid 3.0, 1 per cent., aqueous solution of gentian violet 3.0, and distilled water 300.0.

The little device introduced by Dr. Sterling Bunnell of San Francisco (*J. Am. Med. Assoc.*, Aug. 13, 1910, p. 596), for preventing leakage from the pipette after taking blood at the bedside and while carrying it to the office for examination, may be recommended.

In making the red count after the corpuscles have settled to the bottom of the counting-chamber it is important to follow the rule that all corpuscles within and upon the left and upper border of a square belong to that square. To avoid mistakes from uneven distribution of the corpuscles it is well to repeat the red count in two or three preparations, the ampulla of the diluting tube being re-shaken each time; especially in severe anæmias, where the corpuscles are very few in number, it is well to control the counts carefully.

The *red blood count* gives an accurate idea of the extent of diminution of red cells (*oligocythæmia*), or increase in red cells (*polycythæmia* or *polyglobulia*) which may exist, while the *white blood count*, if abnormal, determines the exact degree of diminution of white cells (*leucopænia*) or increase of white cells present (*hyperleucocytosis*). Should a hyperleucocytosis be demonstrated it is very desirable to proceed to the differential count (*vide infra*) of the white corpuscles in the dried and stained specimens in order to determine whether one is dealing simply with a polymorphonuclear leucocytosis, say of some pyogenic infection, with the eosinophilia of a trichinosis, or with a true leukæmia; the differential count, informing us as it does of the exact varieties of white corpuscles present and their relative proportions, is indispensable for precise diagnosis in such cases.

### III. ESTIMATION OF THE AMOUNT OF HÆMOGLOBIN

The best instrument for clinical purposes is, in my opinion, *Sahli's modification of Gowers's hæmoglobinometer*. It consists, as is well known, of a solution of hæmatin of known strength contained in a hermetically sealed glass tube. In the pipette accompanying the instrument 2.0 c.c. of blood are collected and blown out into a calibrated tube, the pipette being washed out with a few drops of tenth-normal hydrochloric acid solution and these washings

added to the blood. The hydrochloric acid changes the red hæmoglobin of the blood into brown hæmatin chloride. To the blood thus treated water is now cautiously added, drop by drop, until the color of the dilution is identical with that of the fluid in the control tube. One reads off on the scale the amount of hæmoglobin present in the blood under examination in relation to the normal hæmoglobin-content. When making the readings the two tubes are placed against a background of milky glass, since color tints are more exactly discriminated with such a background. In using the hæmoglobinometer of Sahli one should make sure that it has been correctly standardized, since as they come on the market the readings may be too high or too low; if incorrectly calibrated the degree of error should be ascertained by comparison with an accurately standardized instrument and all readings correspondingly corrected.

Two other methods of estimating hæmoglobin are in common clinical use. I refer to the hæmoglobin scale of Tallquist and the Fleischl-Miescher hæmoglobinometer.

Many physicians make use of *Tallquist's scale*, which consists of a series of red colors, the values of which correspond to various percentages of a solution of a normal amount of hæmoglobin. One saturates a piece of filter paper with a drop of the blood to be examined, allows it to dry, and afterwards compares it with the colors of the scale. The readings thus obtained are usually too high, as is shown by control with accurate instruments, though as a rough and ready method the busy practitioner may find it of some value.

The *Fleischl-Miescher hæmoglobinometer* is a very good instrument but is somewhat less convenient than the Sahli instrument above referred to. In it one compares the color of a small quantity of blood diluted to a definite degree with the colors of various thicknesses of a glass wedge containing purple of Cassius. A little blood (0.05 or 0.1 c.c., or 2 minims) is taken up by the minute pipette which goes with the instrument and diluted 200 or 400 times with a 0.1 per cent. solution of sodium carbonate. The examining-chamber is divided into two halves by a median septum and has a glass bottom. One-half of the chamber contains the diluted blood; the other half is filled with distilled water. This chamber is placed upon a stage with a hole in its centre, so arranged that the colored

wedge can be moved back and forth beneath the half of the chamber containing water. By means of a reflector the fluids in both halves of the chamber are illuminated (with artificial light—not daylight) and the wedge is shoved back and forth until the water above it assumes the same tint as that of the diluted blood. One reads off on a scale the percentage of the normal amount of hæmoglobin present in the blood under examination.

**COLOR-INDEX OF RED CORPUSCLES.**—After one has counted the number of red blood-corpuscles in a cubic millimetre of blood and has estimated the hæmoglobin by one of the methods above described, it is easy to calculate the relation of the amount of hæmoglobin to the number of corpuscles, or, in other words, to determine an index of the average individual corpuscular content of hæmoglobin. This relation is known as the *color-index* of the red blood-corpuscles; thus, if we make the normal red count of 5,000,000 to the cubic millimetre equal 100, and similarly the normal amount of hæmoglobin (14 grammes in 100 c.c. of blood) equal 100; then the relation of the hæmoglobin-content to the number of red cells in normal blood will be as 100:100 or as 1:1, and the color-index is said to be 1. When the amount of hæmoglobin is diminished to a greater extent than the number of red corpuscles the color-index will be smaller than 1, while if the number of red corpuscles is more reduced than the hæmoglobin-content the color-index will be greater than 1. This color-index is usually increased in the so-called pernicious anæmia, while it may be markedly decreased in chlorosis and in some secondary anæmias.

#### IV. A STUDY OF THE FORMED ELEMENTS OF THE BLOOD IN DRIED AND STAINED PREPARATIONS

This method has come more and more into use as an aid in general practice; indeed it is indispensable for the exact diagnosis of many of the blood diseases. The blood is spread in thin layers on absolutely clean slides or covers. Some practice is required in getting satisfactory smears, but the results reward the pains. The smears are allowed to dry in the air and are afterwards fixed in heat or by some fixing fluid. For the details of fixation by heating the article by Dr. R. S. Morris (*Jour. Am. Med. Assoc.*, Aug. 6th, 1910, p. 501) will be found helpful, and in his article full directions



for preparing the Ehrlich triple stain and using it will be found satisfactorily given.

Except when staining by Ehrlich's method, instead of fixing by heat one may fix the cover slips by allowing them to lie for from 10 to 15 minutes in equal parts of absolute alcohol and ether, or for from 3 to 5 minutes in absolute methyl alcohol, or for five minutes in chemically pure acetone. Many of the stains now employed, however, do not require previous fixation, since the staining fluids themselves fix and stain simultaneously.

Since the important studies of Ehrlich, we have known that various parts of the white and red corpuscles have different chemical affinities for acid, basic, or neutral dyes; thus, for example, all nuclei stain with basic dyes and are therefore said to be *basophile*. In the protoplasm of many of the white corpuscles are granules; some of these granules stain especially well in basic dyes (*basophile granules*); others preferably with acid dyes (*oxyphile*, *acidophile*, or *eosinophile granules*), and still others stain well with neutral dyes (*neutrophile granules*).

For practical diagnosis it is desirable to use a combined stain (panoptic stain) which will differentiate these substances which present elective affinities. The smears, after they have been stained, are mounted and are studied under an oil-immersion lens with an Abbé condenser. If one were to use only one stain for practical clinical work he would do best to choose one of the so-called eosinate of methylene blue solutions or Ehrlich's triacid stain, the latter being the best stain for the neutrophile granules, the former bringing out the mast cells and the eosinophile granules better. If one desires to study nuclei in particular or is making an especial study of malarial or other parasites in the blood, Giemsa's modification of the Romanowsky stain is best. The *eosinate of methylene blue* is used in various forms (Jenner's stain, May-Grünwald stain, etc.).

It is best for the practitioner to buy his stains ready made rather than to experiment with the solutions, unless he has a good deal of time at his disposal and is especially interested in their preparation. The methylene-blue eosinate, such as Jenner's stain, can be bought in powder or in solution or in tablet form; the latter is very convenient, one tablet dissolved in 10 c.c. (2.7 fluidrachms) of methyl alcohol making a stain ready for use.

Smears are immersed for three minutes in the dye of full strength and then for 10 or 15 minutes in equal parts of the dye and distilled water. They may then be washed with distilled water, dried, and mounted in neutral Canada balsam (Jenner), or they may be immersed for from 5 to 10 seconds in the dye of full strength and then placed in distilled water to which a few drops of the dye have been added and allowed to remain there until some of the blue color has been extracted and the red begins to appear. As soon as the preparation has assumed a light red color it is pressed between folds of filter paper, then dried in the air and mounted (May-Grünwald). In smears stained with this dye the red corpuscles stain red, nuclei blue, basophilic granules blue or bluish-violet, acidophile granules bright red or rose color, neutrophile granules a feeble violet-red or red, blood-platelets grayish-violet, and non-granular protoplasm bluish.

Ehrlich's *triple stain*, best prepared according to the directions given by Morris, yields beautiful preparations after fixation of the smears with heat. The smear is covered with the stain, which is allowed to act for from 5 to 10 minutes, and then quickly washed, dried, and mounted. There is no better method of differentiating the leucocytes, though for studying changes in the red blood-cells and in the nuclei and for searching for blood parasites it is not suitable. In Ehrlich's triple stain red corpuscles stain orange, nuclei green, eosinophile granules bright red, and neutrophile granules purplish-violet. The nuclei of the two kinds of nucleated red blood-corpuscles stain very differently (nuclei of normoblasts intensely green, nuclei of megaloblasts feebly green).

In the *Romanowsky stain* (especially in the much more satisfactory stain *as modified by Giemsa*) the active principle is the reddish-violet dye—methylene azure. The ready-made preparation of Grübler is the best to use. Smears are fixed for from 2 to 5 minutes in methyl alcohol, dried, and then immersed in 1 c.c. (20 minims) of distilled water containing one drop of the dye, left there for from 10 to 30 minutes, washed in distilled water, dried, and mounted. On account of its selective affinity for chromatin this azure stain of Giemsa is most useful in demonstrating malarial parasites and other protozoa in the blood and also for staining the *Spirochaeta pallida* of syphilis. The Leishman-Donovan bodies of kala-azar are

also demonstrable in smears of the splenic juice. In America, similar stains devised by Hastings, Wright, and Wilson are much in vogue.

In Giemsa's stain the red corpuscles take a red or grayish-green tint, the nuclei of leucocytes and nucleated red corpuscles stain red or reddish-violet (nucleoli blue); of the various granulations, the eosinophile take a bright red tint, and the neutrophile stain feebly violet, while in the lymphocytes certain granules known as azure granules, violet in color but not demonstrable by other methods, may be brought out. The protoplasm of the lymphocytes, which has a basophilic tendency, stains blue. This Giemsa stain yields exquisite pictures of all nuclei, of the lymphocytes and of granular red corpuscles, but for the general practitioner its main service will lie in the satisfactory pictures it yields of malarial parasites.

In these dried and stained specimens one studies especially the white corpuscles, but some facts of importance regarding the red cells are also ascertainable. In the first place, *nucleated red cells*, if present, are easily discovered. Small nucleated red cells about the size of an ordinary red corpuscle having rather small nuclei which stain intensely in basic dyes are known as *normoblasts*; they may appear in the blood in large numbers (*blood crises*) after hemorrhage or in severe secondary anæmias and sometimes in pernicious anæmia. In the latter disease large nucleated red blood-corpuscles with feebly staining nuclei (*megaloblasts*) are met with; they correspond to the nucleated red cells of early embryonic life and their presence in the blood in adult life is always of grave import, indicating that the organism has called upon the last reserves at its disposal.

In the anæmias where the bone-marrow is capable of regenerating red corpuscles, in addition to these nucleated red cells certain *immature, non-nucleated forms* appear. These are recognizable by the fact that the red corpuscle takes on some of the methylene-blue stain (so-called *polychromasia* or *polychromatophilia*). Another appearance occasionally met with, especially in lead-poisoning, is the presence of *basophilic granules* in the red corpuscles.

**DIFFERENTIAL COUNT.**—In studying the white corpuscles in stained preparations it is necessary to make a so-called differential count with an oil immersion lens and a mechanical stage. Passing over field after field and making sure to examine the periphery

of the smear as well as the more central parts, one counts from 300 to 500 white cells, placing a stroke in a separate column for each white corpuscle of a particular variety met with. One can then easily calculate the relative percentages of each kind present in the blood, and by utilizing the total white count one can also get a tolerably accurate estimate of the absolute number of each variety of white cell present in a cubic millimetre of blood.

Normal blood containing say 7000 leucocytes per cubic millimetre will present in the differential count of the leucocytes numbers approximately as follows:

1750 lymphocytes .....	25.0 per cent.
4970 polymorphonuclear neutrophiles .....	71.0 per cent.
70 large mononuclear and transition forms .....	1.0 per cent.
175 eosinophiles .....	2.5 per cent.
35 mast cells .....	0.5 per cent.

In normal blood no *myelocytes* (large granular mononuclear cells) are visible, but in the myeloid leukæmias these cells may be present in enormous numbers.

The relative percentages above given vary within somewhat narrow limits in health. Any outspoken deviation, however, is to be regarded as abnormal.

In the ordinary *hyperleucocytoses*, where the leucocyte count is over 9000 per cubic millimetre and may reach 12,000, 20,000 or over 100,000 per cubic millimetre, the increase in the cells is due chiefly to the presence of an abnormally large number of polymorphonuclear neutrophiles. Slight physiological leucocytoses are met with in digestion, during pregnancy, after exercise, overheating, etc. In pathological conditions polymorphonuclear leucocytoses are seen: (1) in the pyogenic infections, due to streptococci, staphylococci, pneumococci, meningococci, etc., in diseases like septicæmia, pneumonia, erysipelas, tonsillitis, arthritis, endocarditis, meningitis; and (2) in the secondary anæmias (due to hemorrhage, tuberculosis, or neoplasm).

When the total white count is diminished (*leucopænia* or *hypo-leucocytosis*) the finding is often just as important for diagnosis as its opposite; thus a leucopænia is very helpful in the early diagnosis of typhoid fever, measles, and parotitis. Occasionally a

leucopænia is seen in severe infections of the type which when milder usually give rise to a hyperleucocytosis (e.g., pneumonia, sepsis). High grades of leucopænia are sometimes met with in the form of pernicious anæmia known as the aplastic type.

A marked increase in the percentage of eosinophiles (*eosinophilia* or *eosinophilosis*) in the blood often points to some form of worm invasion (*Trichina*; *Bothriocephalus*; *Echinococcus*), to some skin disease (especially pemphigus, arsenical exanthems and scarlet fever), or to bronchial asthma. Dr. T. R. Brown first pointed out that the eosinophiles may make up more than 50 per cent. of the white corpuscles in the blood in trichinosis.

The large mononuclears and transition forms are often relatively and sometimes absolutely increased (*large mononucleosis*) in tuberculosis, in syphilitic infections, and in typhoid fever and malaria.

An increase of the small mononuclear cells or lymphocytes is known as a *lymphocytosis*. This may be either relative or absolute. A relative increase is common in typhoid fever, in syphilis, and in aplastic anæmias, while great absolute increases are met with in lymphatic leukæmia.

In the *leukæmias* the number of white corpuscles may be so increased as to approach that of the red cells; indeed cases are on record where more white cells than red were present. The differential white count distinguishes two great types of leukæmia: (1) lymphatic leukæmia, and (2) myeloid leukæmia.

In *lymphatic leukæmia* the lymphocytes may make up more than 90 per cent. of all the white corpuscles. The disease may be acute or chronic, the acute cases dying usually within sixty days after the onset. In these acute cases instead of cells of the normal lymphocyte type their mother cells (so-called lymphoblasts) may appear in the blood.

In *myeloid leukæmia* it is not the lymphocytes which are predominantly increased but the cells which have their origin in the bone-marrow especially; namely, the polymorphonuclear leucocytes and eosinophiles and their mother cells; indeed the mother cells (myelocytes) may be far more numerous than the daughter cells. In severe cases the grandmother cells (myeloblasts) are also present in the blood.

In *Hodgkin's disease* and in the *aleukæmic leukæmias* (Blumer)

no marked changes occur in the white-cell count, notwithstanding the extensive hyperplastic changes in the lymphadenoid and myeloid tissues.

#### V. CERTAIN BACTERIOLOGICAL AND BIOLOGICAL EXAMINATIONS OF THE BLOOD

**BLOOD CULTURES.**—In suspected typhoid fever, in paratyphoid infections, and in general septic processes (endocarditis, polyarthritis, septicæmia, pyæmia, etc.) a *blood culture* will often throw light upon the diagnosis and in some instances may be helpful in deciding upon specific therapy (vaccine therapy, antitoxic therapy). About 2 c.c. (30 minims) of blood are withdrawn from the median vein at the bend of the elbow (in a syringe which has been sterilized by heat) and placed immediately in an Erlenmeyer flask containing 50 c.c. (2 fluidounces) of either glucose-bouillon, or, if typhoid fever be suspected, sterile ox-bile containing a little salt and peptone, or a mixture of bile and bouillon. The flask is kept in a thermostat at the body temperature for from twenty-four to forty-eight hours and then examined by bacteriological methods for the presence of streptococci, staphylococci, pneumococci, typhoid bacilli, etc. The blood-culture is of the greatest importance in the early diagnosis of typhoid fever. On the average a positive diagnosis of typhoid or paratyphoid infection can be made at least a week or ten days earlier than is possible by the Widal reaction or by any clinical method of examination.

**2. THE WIDAL REACTION.**—In many infectious diseases substances known as *agglutinins*, specific for the micro-organism which causes the infection, appear sooner or later in the blood during the course of the disease. These substances, discovered by Durham and Grüber in the course of their scientific investigations, have come to be of importance in practical clinical diagnosis, thanks to the suggestion of the French observer Widal.

If a drop of fresh bouillon culture (24 hours old) of typhoid bacilli showing active motility be mixed with the serum of a patient suffering from typhoid fever in whom these agglutinins have developed, the bacilli, at first more or less evenly diffused and freely suspended in the fluid, become so altered that their motility ceases and they aggregate in clumps (*agglutination*) and fall to the bottom of the fluid. Normal human blood-serum possess slight agglutinat-

ing power and may show it in dilutions of 1-10, but in the second week of typhoid fever and later the serum of the patient will often cause agglutination in dilutions of from 1-100 to 1-1000. Owing to the necessity (1) of keeping a reliable typhoid culture going every day for the test, and (2) of a good deal of practice in observing the reaction, the test, though highly valued by the practitioner, is seldom made by him personally, since thanks to the number of private bacteriological laboratories and to the laboratories of municipal health offices it is now easy for the general practitioner to have his Widal tests made for him at little or no cost.

The mere qualitative reaction is no longer sufficient for exact diagnosis. It is necessary to make *quantitative determinations* of the agglutinative power; in other words, to determine at what extent of dilution the agglutination ceases. The reason for this quantitative work lies in the fact that all bacteria of a group to which a given bacterium belongs may be agglutinated by the serum of a patient suffering from an infection with any member of that group (*group agglutination*). For example, typhoid bacilli, paratyphoid bacilli, and colon bacilli belong to one group. The serum of a typhoid patient may agglutinate typhoid bacilli very powerfully (even in dilutions of 1:600); it may also agglutinate paratyphoid bacilli, though less markedly (dilutions of 1:100), and it may agglutinate colon bacilli feebly (dilutions of 1:20). The same serum might agglutinate a cholera vibrio (belonging to a different group) in dilutions of 1:10. Normal blood-serum might agglutinate typhoid bacilli, paratyphoid bacilli, colon bacilli, and cholera vibrios, all to about the same extent (very feebly in dilutions of 1:10). If the fever under study clinically were due to the paratyphoid bacillus a stronger agglutination would occur with this bacillus and a feebler with the typhoid bacillus. Great care has to be exercised, however, in working with paratyphoid bacilli since some strains agglutinate almost spontaneously.

Attempts have been made to use dead typhoid bacilli for agglutination in order to avoid the necessity of keeping living cultures constantly going. This is the basis of *Ficker's method* (a suspension of typhoid bacilli killed with formalin now on the market), and of other similar preparations.

In taking the blood for a Widal test a small amount is sucked up into a glass tube, the ends of which are sealed with hot sealing-

wax. After the blood has coagulated the serum is drawn off by means of a capillary tube and dilutions of serum of various strengths made. These dilutions are mixed with the Ficker reagent or with the living culture. Controls of normal human serum in equal dilutions should always be observed.

Too much has been expected of the Widal reaction by many practitioners. The following facts should be kept in mind:

*a. A negative reaction does not necessarily mean that no typhoid or paratyphoid infection is present. In some cases no agglutinins are formed throughout the whole period of the fever; in a few cases they appear in the blood a few days after the patient goes to bed. The method is far less valuable for diagnosis in the early stages of the disease than the blood culture.*

*b. A positive reaction does not necessarily mean that an active typhoid infection is at the moment going on in the body. The Widal reaction may remain positive for weeks or even months in convalescence after the typhoid fever is over; it may be present in certain sequelæ of typhoid fever due to local infections with the typhoid bacillus (chronic cholecystitis, typhoid bone infections, etc.); in a certain number of chronic typhoid-bacillus carriers the Widal reaction is positive.*

Aside from the diagnosis of typhoid and paratyphoid infections, agglutinin reactions have not been of especial value in clinical diagnosis. A similar principle is used, however, in the *identification* of cholera bacilli grown from the stools of suspected cases of cholera, of the meningococcus grown from the cerebrospinal fluid, and of bacilli grown from the stools of cases of bacillary dysentery (Shiga bacillus; Flexner bacillus). Here the blood-serum of animals artificially immunized against these specific organisms is used to identify the specific nature of bacteria found in human beings—just the opposite of the Widal reaction in typhoid fever, where a known bacillus is treated with the serum of a patient, suspected to be infected with that bacillus, in order to see if he has developed an agglutinative immunity against it.

3. THE WASSERMANN REACTION.—The Wassermann reaction has recently attracted a great deal of attention and the sero-diagnosis of syphilis has proved to be of much practical importance. The method depends upon an application of the principle of complement-fixation which we owe to Bordet and Gengou. Fully to



understand it, it is desirable to have some knowledge of the side-chain theory of Ehrlich and to know what is meant in that theory by the terms amboceptor and complement. It would take too long to give even a brief description of this theory here, but, in a few words, it may be recalled. According to Ehrlich's view, in certain forms of immunity substances appear in the blood known as *amboceptors* because they appear to possess two chemical groups, one for uniting with the poison of the specific micro-organism (*antigen*) against which the immunity has developed, the other for uniting with a constituent of the blood known as the *complement*. Though the amboceptor may unite with the antigen in the absence of complement, it cannot exert its effect upon this until complement is added and unites with its other group. Amboceptors are specific bodies, that of typhoid immunity, for example, being different from that of syphilitic immunity, that of immunity against red corpuscles being different from that of immunity against the pneumococcus, etc. Complement, on the other hand, is present in normal blood, as well as in diseased blood, and appears to be able to unite with different amboceptors; in other words the toxophile or antigenophile groups of the amboceptors are different and specific, the complementophile groups may be identical. In immunity against a foreign variety of red blood-corpuscles the hæmolytic substances consist of specific hæmolytic amboceptors (*hæmolysins*) which unite through one of their groups with the corpuscles, and through the other group with the non-specific normal complement. When the three are united hæmolysis occurs. Similarly in bacteriolytic immunity, the bacteriolysis occurs through the mediation of the amboceptors (*bacteriolysins*) which unite through one of their groups with the bacteria (antigen) and through the other group with the complement.

In the Bordet-Gengou *phenomenon of complement-fixation*, use is made of the principle that on mixing an antigen with its specific amboceptor a union takes place which has an extraordinary affinity for any free complement present and tends to use this up by combining with it, while amboceptors alone unattached to antigens have less affinity for complement. If one, therefore, mixes (A) a serum which is suspected to contain specific amboceptors but is deprived of complement by heating it to 56° C. (133° F.) with (B) the antigen corresponding to these specific amboceptors and then adds (C) fresh complement (in the form of unheated serum) and allows the

mixture to stand long enough for union to take place, one can prove whether or not fixation has taken place (and so whether the specific amboceptors were really present for the antigen under consideration) by adding a hæmolytic serum (rendered inactive by heating) and some red blood-corpuscles; if the complement has united (complement-fixation, sometimes erroneously called complement-deviation) no hæmolysis will occur; (D) if the complement has not united it will reactivate the hæmolytic serum and hæmolysis will occur.

The five substances necessary for carrying out the complement-fixation reaction are: (a) the antigen; (b) the amboceptor; (c) the complement; (d) the hæmolysins; and (e) red blood-corpuscles.

The (a) *luetie antigen* is best prepared from the livers of children dead of hereditary syphilis. The (b) *amboceptor* is the specific antibody, the presence of which one tests for in the blood of the patient suspected to be suffering from syphilis (using serum of the patient heated to 56° C. (133° F.) for half an hour in order to destroy its complement). The (c) *complement* is derived from the normal blood-serum of guinea-pigs. The (d) hæmolysins (inactive hæmolytic serum) are secured by heating the blood-serum of a rabbit which has been immunized against the red blood-corpuscles of a sheep, and (e) the *red blood-corpuscles* used in the experiment will therefore be the normal red blood-corpuscles of the sheep, carefully washed free from their serum and suspended in salt solution.

In a test-tube one mixes the antigen, the suspected serum of the patient and the complement and allows them to stand for an hour in the thermostat in order that union may take place; the hæmolytic serum and the washed sheep's corpuscles (the two together making a so-called "hæmolytic system") are next added, the test-tube carefully shaken and again placed for two hours in the thermostat alongside of controls made with normal serum.

Where all the red corpuscles are dissolved the reaction is said to be complete; if there remain any turbidity, the reaction is said to be incomplete. When there is no hæmolysis at all the reaction is entirely negative. Here again quantitative relations are important and the laboratory workers have devised methods for establishing the degree of immunity which exists. It is wholly impracticable for the general practitioner to make the tests himself. He must be content with sending specimens of blood to special labora-

tories for the examination, and, even there, the results are reliable only when men who are carefully trained and who have had considerable experience with the technic do the work. Much of the conflicting testimony regarding the value of the Wassermann reaction has been due to imperfect work.

A modification of the Wassermann reaction which makes the technic simpler is that introduced by Noguchi of the Rockefeller Institute. In Noguchi's hands the method appears to yield reliable results, but further work must be done before we can decide the liability to error in the hands of less skilled workers.

In the clinic in Baltimore in which I work, Dr. Clough, Dr. Guthrie, and Dr. Austrian, who make the serological tests, have relied chiefly upon the original Wassermann reaction.

Experience teaches that the reaction is fairly constant in patients in whom active syphilis exists (even decades after the original infection). The reaction may be negative during the primary lesion and occasionally early in the secondary stage or in cases that have recently been subjected to mercurial treatment or to Ehrlich's *therapia magna sterilizans* with the arsenical preparation, "606."

In patients suffering from general paralysis the blood-serum nearly always yields a positive Wassermann reaction and the cerebrospinal fluid is also positive in a large proportion of the cases, thus demonstrating the syphilitic nature of this disease. In tabes dorsalis only about half the cases yield positive results, a fact which has led some back to the opinion that tabes may sometimes be due to syphilis and sometimes to other causes. We have been struck in Baltimore with the frequency of positive reactions in patients suffering from aortic aneurism, particularly when the aneurism appears in early or middle life, a corroboration of the view that most of these aneurisms are due to a syphilitic aortitis. In many cases suffering from aortic insufficiency the Wassermann reaction is positive.

With the aid of the Wassermann reaction many mild cases of lues can be recognized which would otherwise be entirely overlooked. It is especially valuable in making diagnoses in families in which one member is known to be infected (diagnosis of conjugal lues and of congenital lues).

The reaction may also be employed as a satisfactory guide in antiluetic therapy. Every luetic patient should be tested at intervals until, finally, the Wassermann reaction as a result of adequate treatment becomes permanently negative.

## FUNCTIONAL TESTS OF CARDIAC EFFICIENCY

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THE natural outcome of studying the heart by instrumental methods was the development of a number of quantitative tests of cardiac efficiency. Each of these tests was based upon some specific reaction of blood-pressure or pulse-rate as the result of some definite effort, change of position, etc., in which the reaction of the weakened or diseased heart was found to differ from the normal. Since all severe effort sooner or later will give rise to symptoms on the part of the patient which serve as some guide to the condition of his circulation, the importance of these functional tests by instrumental methods depends largely upon whether the changes in pulse or blood-pressure characteristic of the weakened heart occur before the appearance of symptoms or whether the symptoms appear before the characteristic changes in pulse or blood-pressure.

The principal functional tests that have been employed are: (1) The change in pulse rate which occurs when the patient stands up after lying down. (2) The change in pulse-rate which follows the simultaneous contraction of muscles which antagonize one another. (3) The change in blood-pressure upon compressing the femoral arteries. (4) The change in blood-pressure during exercise.

1. When a normal individual rises from the reclining to the standing posture his pulse-rate becomes accelerated, but the amount of acceleration is usually less than twenty beats per minute. In persons with failing hearts this change of rate upon rising is more than twenty (Stephens).

In many cases this test furnishes valuable information, but there are numerous individuals in whom it fails absolutely to establish a criterion. For convenience these may be grouped as follows: (a) Persons with normal hearts, in whom the psychic element plays a considerable rôle, whose heart rate may increase inordinately upon the change of position. (b) Persons with enteroptosis, upon

whose circulation gravity plays an unusually large rôle, but whose hearts are nevertheless not especially weakened. (c) Persons with chronic myocardial changes, especially in the sinus region of the auricle, whose hearts are already responding with as many impulses per minute as the muscle fibres of this region are capable of generating. This is especially common in cases of permanent absolute arrhythmia.

2. Another test based upon change in pulse-rate has been introduced by M. Herz, and is known as the Self-checking Test (Selbst-hemmungsprobe). The patient is made to sit down until the pulse-rate has become constant. He is then made to contract all the muscles of his hand and forearm with all the force possible, and to flex and extend the forearm very slowly, focussing all his attention upon the movement, and attempting to antagonize his own movements with as much force as possible. Herz states that in healthy persons the pulse-rate is unchanged by this manœuvre, whereas in persons with weak hearts it becomes 5 to 20 beats less per minute. Cabot and Bruce have found that Herz's test holds in a certain number but they are unwilling to subscribe to it as a general rule, and I have myself met with a certain number of perfectly strong and healthy persons who give Herz's pathological reaction. There seems, indeed, little physiological basis for so absolute a classification upon this reaction, which depends quite as much upon the degree of irritability of the vagus as it does upon the cardiac output and cardiac vigor. The frequency of exceptions to Herz's rules is therefore not surprising.

3. Marey in 1881 found that if both brachial and both femoral arteries of the patient were compressed by an attendant, the patient's blood-pressure would be found to rise if his heart were vigorous, to fall if his heart were failing. This reaction has recently been revived by Katzenstein who compresses the two femorals only. Katzenstein obtains the following results:

Subject	Blood-pressure	Pulse rate
Normal individuals.....	Rose 5-15 mm.....	Fell
Compensated heart lesions..	Rose 15-40 mm.....	Unchanged or fell
Slight cardiac insufficiency..	Unchanged.....	Unchanged or rose
Very weak hearts.....	Fell.....	Fell

This test is founded upon the results of experiments which serve as a basis for the other functional tests also, which also serve to mark out their limitations.

Romberg and Hasenfeld have shown that if the descending thoracic or the abdominal aorta be clamped or compressed the blood-pressure rises exactly as it does upon compression of the femoral arteries in man. In certain animals whose hearts were weakened to an extreme degree the blood-pressure fell instead of rising upon compressing the aorta. However, they found that the rise of pressure occurred even in dogs with experimental aortic insufficiency, and Welch and Von Stejskal have found that it occurs in dogs whose heart muscle has been injured by diphtheria toxin and by poisoning with phosphorus. Upon this basis alone, therefore, there is in these experiments no sharp difference between the reaction of normal hearts and that of hearts whose myocardium has been injured to a considerable extent.

De la Camp, however, was able to demonstrate such a difference. He examined with the orthodiagraph the hearts of normal dogs before and after running on a treadmill to the point of exhaustion as well as those of dogs which had been poisoned with phosphorus. The hearts of the normal dogs either became smaller or underwent no change whatever as the result of overwork. The hearts of the dogs with phosphorus poisoning became dilated as the result of the same exercise, although, as previous experimenters had shown, such hearts were still capable of reacting with a rise of blood-pressure. The exact relations which acute cardiac dilatation bears to the changes in blood-pressure as the result of strain has been very clearly shown in some experiments which I made in collaboration with Dr. Percival D. Cameron, tracings of which are shown in Fig. 1.

Fig. 1 shows the effect of clamping the thoracic aorta in a dog with a strong heart upon the volume of the heart and the blood-pressure in a dog with a vigorously beating heart. Immediately after clamping, the blood-pressure is seen to rise, the maximal pressure more than the minimal, and the systolic output of the heart increases. Nevertheless, the heart dilates under the strain. In the animal with the strong heart this dilatation is only momentary, for in less than a minute it may be seen to have pumped out the entire excess of blood, and to have reached its original volume. Indeed,

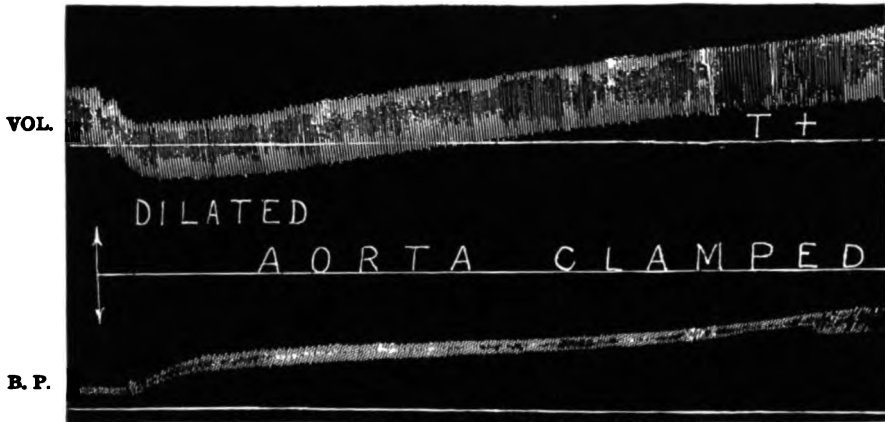
after another minute as a result of the increased output at each systole as well as of an improved tonicity, the volume of the heart has become smaller than before the strain was put upon it. Blood-pressure and pulse-pressure are thus seen to rise and systolic output and tonus to increase from strain thrown upon the vigorous heart.

Fig. 2 represents the same experiment performed upon a dog whose heart was in a weakened but not an extremely weakened condition. Clamping of the aorta here causes a rise of blood-pressure but this is transitory and is followed by a fall. The heart dilates at once and though its output at each beat soon returns to normal, it remains dilated throughout the curve. The weakened heart responds to the effort with a transitory rise of blood-pressure followed by a fall, and the heart itself remains dilated. With or without change of blood-pressure the tonicity of the heart muscle is impaired. The extreme degree of cardiac weakness is indicated diagrammatically in Fig. 3, curve 3. The heart not only dilates at once under the strain but the dilatation increases progressively until the heart becomes overfilled to the point at which effective contractions become impossible, and death ensues.

The conditions observed in carrying out the Marey-Katzenstein compression test are exactly similar to those of this experiment upon animals. Hoke and Mende, who have tried it in a large series of cases, find that though the results hold true in general, they are often uncertain and sometimes incorrect. On the other hand, in very severe cases, the test is so severe that its use becomes dangerous. These clinical results could, indeed, be prophesied from the experiments. It will be seen (Figs. 1 and 2) that there is a considerable borderland between the vigorous and the weakened heart, in which both bring about a rise of blood-pressure, but in which the volume of the vigorous heart is normal or smaller than normal, while the weakened heart has dilated, *i.e.*, the residual blood in the heart is increased and the tonicity of the heart muscle is diminished. The reaction of blood-pressure therefore gives no index of this condition in the heart muscle, and to rely upon it alone is accordingly deceptive.

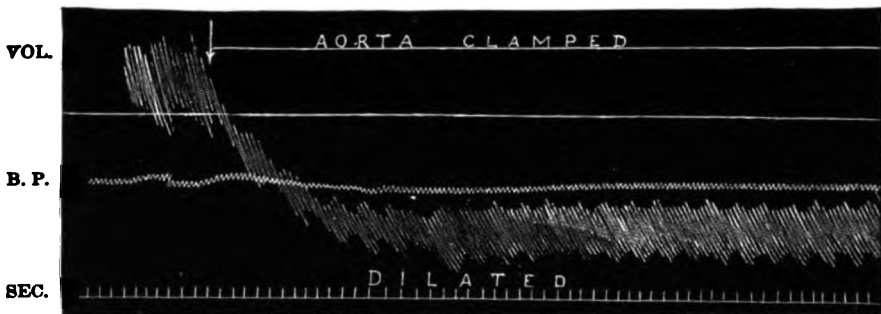
A moderate grade of dilatation of this character can rarely be made out by percussion, and not always by means of the orthodiagraph; yet the strain is entirely too severe to be imposed upon the

FIG. 1



Effect of strain upon the dog's heart whose tonicity is good. Volume curve (VOL.) and blood-pressure curve (B. P.) of an animal whose heart is in good condition. Descending thoracic aorta clamped at the moment indicated by the arrow. Momentary dilatation followed by a diminution in size. The heart becomes smaller than before the clamping. Tonicity is increased (T +). Blood-pressures maximal and minimal are also increased.

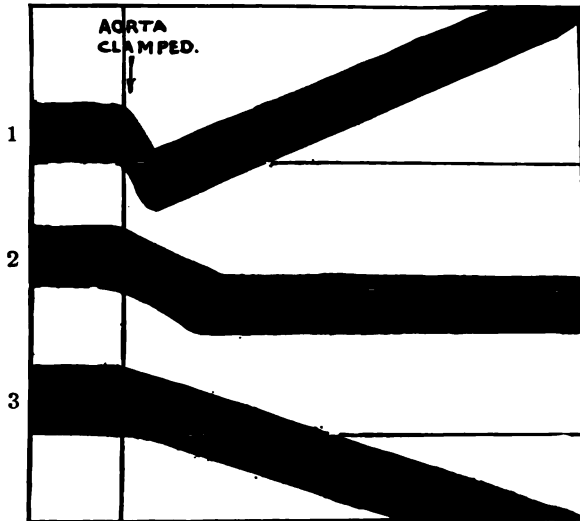
FIG. 2.



Volume-curve of a dog whose cardiac tonicity is low. Clamping the aorta is followed by permanent dilatation and only a slight momentary increase in blood-pressure. The systolic output is diminished, owing to inability of the heart to force the usual quota of blood against the increased resistance. SEC., time-markings in seconds.

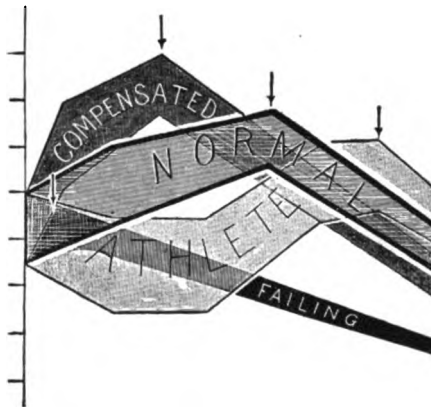


FIG. 3.



Effect upon the volume of the dog's heart produced by clamping the descending thoracic aorta. Ascent of curve—diminution in volume; descent—dilatation. 1, normal and vigorous heart—strain followed by diminution in volume; 2, slightly weakened heart with decreased tonicity—strain followed by persisting dilatation; 3, very weak heart with greatly diminished tonicity—strain followed by progressive dilatation and death.

FIG. 4



Effect of prolonged exercise upon the blood-pressure of persons with various degrees of cardiac efficiency. The ordinates represent blood-pressure—the upper lines of the curves representing the maximal (systolic), the lower curve minimal (diastolic) pressure. The shaded area represents the pulse-pressure. The abscissæ represent time. The arrows indicate the onset of exhaustion. COMPENSATED, compensated heart lesions; FAILING, broken compensation with heart failure.

patient with equanimity. On the other hand, it must be added that the patient often experiences symptoms of discomfort pointing to circulatory inadequacy before the blood-pressure changes have become characteristic, so that the symptomatic reaction of the patient is more definite than the quantitative changes in blood-pressure.

4. Masing, and later Gräupner and a host of other observers have aimed to test the efficiency of the heart by the changes in blood-pressure occurring as a result of exercise. Taking certain definite forms of exercise, such as the rhythmic raising of weights by the feet or the hands, it is found that the normal blood-pressure rises during the exercise and falls immediately afterwards. The amount of rise is more or less proportional to the effort made by the subject.

For chest-weight exercise it is found that the maximal blood-pressure of normal individuals may undergo a rise of from 10 to 30 mm. of mercury, while that of arteriosclerotics and persons with moderately weakened hearts the rise of pressure may be 40 to 60 mm. and may outlast the period of exercise. Baur makes use of a stationary bicycle instead of weights and has found that in normal individuals the blood-pressure rises 5 to 10 mm. of mercury, while in persons with cardiac insufficiency there is a fall of 5 to 10 mm. The exercise test is indeed the most valuable of all, though considerable caution must be observed not only in the execution of the exercise, but also in the interpretation of the findings.

In the first place it must be recalled that since the blood-pressure change is proportional to the effort on the part of the subject, the amount of change that occurs depends not only upon the condition of his heart but upon the condition of his muscles. A person with flabby muscles but with a strong heart may according to this test give evidence of as great circulatory disturbance as is given by one with strong well-trained muscles but a somewhat weakened heart. Moreover, just as in the compression test, the blood-pressure figures give no index of the degree of dilatation or of impairment in tonicity of the heart.

On the other hand, the reaction of very strong persons to exercise is quite different, but leads to another ambiguity in the records of the tests. Dr. G. A. Gordon as well as Professors Dawson and Eyster have shown that in trained athletes the blood-pressure falls, instead of rising, at the commencement of mild exercise, and this

fall may last for a considerable period. The reaction of the strongest hearts therefore resembles closely the reaction of the weakest.

The effect of continued mild exercise upon the blood-pressure in hearts of various degrees of strength illustrating these relations is shown in Fig. 4.

It must be seen therefore that all of these so-called functional tests based upon mathematical changes in pulse and in blood-pressure present many uncertainties and ambiguities; and that in the search for characteristic figures it is very easy to overstep the limits of the patient's strength.

This statement must not be construed as an objection to functional tests of cardiac efficiency as a whole, but merely to those based upon numerical changes. As has been seen above, these tests do not take into account the changes in residual blood and in cardiac tonicity. On the other hand, nature has given us a far more delicate index in the sensations and appearance of the patient himself. The acceleration or labored character of the breathing, holding of the breath, dilatation of the nostrils, drawing in of the corners of the mouth, the appearance of dusiness or pallor in the patient's cheeks, or the onset of sweating or palpitation represent signs which appear earlier, are more easily observed, and are less ambiguous in their significance than are the numerical changes in blood-pressure or pulse-rate.

The character of the exercise used as a test should be varied to suit the occupation of the patient and should represent some form of exercise to which his muscles are already trained and which he will be compelled to carry out in the course of his daily life after passing out of the active care of the physician. Thus, for an agent this would be walking or stair-climbing; for a laborer it might be the lifting of weights; for the soldier it might be the running of races. By taking as a standard some arbitrary amount of an exercise to which the patient is accustomed, and observing from day to day or week to week the amount of exercise which the patient can take before the slightest symptoms manifest themselves, it is possible for the physician to form a fair estimate not only of the general cardiac efficiency and the improvement of the patient, but also of the place which he will be able to assume in the world which he is to re-enter.

For patients confined to bed for cardiac weakness the ordinary

functional tests are too severe. For them Herz's self-checking test is well adapted, especially if the observer's attention is focussed upon the condition of the patient as well as on his pulse-rate. Still more satisfactory is the use of an exercise which consists of extending the arms directly forward from the shoulders, then swinging them backwards and outwards until they form a line with the shoulders, and repeating this movement as rapidly as possible. Although this exercise can be made for the moment very severe, the position of the patient and the muscles used in the exercise are such that even in the weakest its effects usually subside within less than a minute. I have never known it to leave any residual discomfort such as may be brought about by other exercises or postures.

Perhaps the most important point in the execution of any form of exercise as a functional test is that the physician must keep his patient's mind diverted from the exercises that he is carrying out, for there is very frequently a strong psychic element in his weakness. It is quite common to find patients who show signs of dyspnoea and apparent exhaustion at the commencement of the exercise, who are nevertheless quite able to carry it out without the appearance of any of these signs when their attention is distracted. This condition was exemplified by a middle-aged woman whom I recently examined who became very short of breath and complained of exhaustion at the very beginning of the arm exercises. However, after I had drawn her into conversation she was able to carry on both conversation and rapid exercise, laughing and joking most of the time, without a catch in her breath or sign of respiratory difficulty, for a period which would have been creditable to a healthy person. Again, the same test was carried out unconsciously by a young man with a mild smoker's heart who complained of palpitation and weakness, but whose daily exercise consisted of a brisk run of several hundred yards which he performed without any difficulty and which was never followed by symptoms. The crux of the problem is summed up in the following anecdote quoted by Friedrich Müller: "Once a young physician, climbing up a steep hill in company with an experienced physician, described to him in rapid conversation his cardiac troubles and palpitations. The elder physician replied: 'You have a nervous heart and no organic heart trouble, else during climbing you would not continue to talk but rather would gasp for breath.'"

It is evident, therefore, that every movement, every exertion made by the patient becomes a functional test of cardiac efficiency when it is properly noticed by the physician, and that those exertions which are carried out spontaneously and unconsciously represent the real strength of the patient. The formal set tests have their place, and a not unimportant one, in clinical observation, but in their interpretation the first rule for the physician should be, "Do not study the chart until you have first obtained all the information that you can by watching the patient."

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# Medicine

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## GANGRENE OF LIMB DURING CONVALESCENCE FROM DIPHTHERIA

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By diligent search in the Libraries of the Royal Society of Medicine and of the Royal College of Surgeons of England, it has been possible to find records of eight cases of gangrene following diphtheria. We do not mean gangrene of the parts near the situation of the disease, such as sloughing of throat or tonsils. We refer to gangrene of parts distant from the disease, such as a limb. That such occurrence is very rare is proved by the fact that Dr. Caiger of the South Western Fever Hospital, London, in seventeen years' work had never heard of a case; besides, by the paucity of records in the literature. In consequence, it struck us that it would be a useful work to collect together with our case the various items of knowledge scattered through the literature making nine cases in all. In connection with this we acknowledge our thanks to Dr. J. D. Rolleston of the Grove Hospital for the details in his letter to Dr. Caiger.

The gangrene in these cases is connected with a definite vascular lesion. In six out of nine cases, the lesion was a definite embolism; in one, it is termed Raynaud's Disease; and in two, the origin is obscure, as the papers, published in Madrid and Venice about 23-34 years ago, are unobtainable. In no less than seven out of the nine cases, the leg was affected; in one, there were wide-spread

gangrenous lesions in addition. In the other case there was a widespread necrotic affection. Thus it would appear that in 6 out of the 9 cases, the gangrene was due to a vascular lesion and that in 3 it was due to a vascular lesion with the addition of an infective or septic character of the embolus.

In only one case, which we record, was the diphtheria treated with antitoxin. The majority of the patients naturally were children.

The origin of these emboli seem to be in the auricles, particularly the left auricle because in only one case was there also a pulmonary embolus. The embolism occurs during convalescence, at the time of reaction from the illness; in one case on the 17th day of the illness. It seems to occur only after a severe diphtheritic attack. Subsequent to the embolism the temperature rose to  $37.7^{\circ}$  C. ( $99.8^{\circ}$  F.) from subnormal, to which level it subsided, rising again in a few days time from septic absorption from the gangrenous part.

It is a curious fact that surgically the femoral or popliteal artery can be ligatured in young subjects without causing gangrene. And yet a postdiphtheritic embolus may cause gangrene! The difference must be due to a thrombus extending from the embolus; and in diphtheria, we would suggest, an arteritis having arisen from endarteritis preventing the dilatation of the branches to form an anastomic circulation; in addition there is the dystrophy of the diphtheritic paralysis and the possibly infective condition of the embolus.

We will give a full account of our case first and then reproduce briefly those obtainable from the literature.

*Diphtheria; threatened cardiac failure; albuminuria; paralysis; gangrene of foot; amputation of leg; recovery.*

The illness began on Nov. 28, 1908, with shivering and vomiting. On the 29th the throat was sore but no membrane was visible. On the 30th, thick membrane on throat. Injected with antitoxin 4000 units; removed to Southgate Isolation Hospital. Dec. 1st, antitoxin 4000 units injected. Dec. 4th, piece of tough, blood-stained membrane  $2 \times 1$  in. syringed from throat. Dec. 5th, vomiting and pains in stomach. Dec. 12th, vomited 4 hours. Threatened with collapse and cardiac failure; pulse could not be felt. Extremities cold; condition much the same for 3 hours. Gradual improvement during night, but could not retain any nourishment. Dec. 13th, still vomiting; all nourishment by mouth stopped except water by spoon occasionally. On 14th, nutrient enema of eggs, milk, and brandy given every 4 hours. Dec. 15th (seventeenth day),

cramp in right leg very acute; patient screaming with agony. Hot flannels and bathing in hot mustard water gave temporary relief. Dec. 16th, cramp at frequent intervals, relieved by laudanum fomentations. Dec. 17th, right foot and leg slightly discolored in patches, giving appearance of bruises. Dec. 18th, patches of discoloration over right foot and leg up to knee more marked; purple in color; the patches over foot and lower part of leg larger and darker than those higher up. *Urine*: very scanty; albumin, 50 per cent. Taking milk from spoon. Dec. 19th, nutrient enemata discontinued. Albumin, 75 per cent.; no vomiting. Dec. 22d, albumin down to 50 per cent. again. Patches of discoloration at upper part of leg beginning to fade and subside, while those over foot and lower part of leg are larger and deeper in color; leg tender. Dec. 24th, albumin none; leg kept wrapped up in wool. Dec. 25th, had a little chicken and vegetable. Dec. 28th, feeding well; 3 eggs daily. Discoloration of leg practically subsided down to below juncture of middle and lower third of leg and foot. The patches over foot and lower part of leg have coalesced so that the foot is of an even purple color. Jan. 1, 1909, complains of pain in foot and leg, keeps the leg flexed and lying on its outer side, resents its being touched. Jan. 3d, blister appeared on the ankle; toes black, dry, and shrivelled. Jan. 4th, coughing after fluid nourishment; voice very nasal; general condition very paralytic. Jan. 5th, very weak; seems to be sinking; great difficulty in swallowing, especially solids. Jan. 7th, gradually improving; but pulse, which has been very rapid all along, still remains so (130 to 160); sleeping well. Jan. 15th, improvement maintained; feeding well; marked line of demarcation at junction of lower and middle thirds of leg; and sloughing rapidly immediately below line of demarcation. Jan. 30th, leg amputated just below knee. Feb. 12th, stump quite healed; pulse normal. Boy in excellent health and putting on flesh. March 15th, discharged.

During the operation, on the forty-sixth day after the embolism and the sixty-first of the illness, the popliteal artery was divided just above its bifurcation. Its lumen was free from clot, it was markedly small and the walls were thin. Hence the embolus was above the bifurcation of the popliteal artery; possibly in the femoral artery, at the bifurcation of the superficial femoral. This view is supported by the clinical observation that on the third day after the embolism, Nov. 18th, the discoloration reached the level of the knee. The discoloration receded from the knee as the anastomotic circulation improved and finally established a line of demarcation three inches above the ankle.

After the amputation, the arteries in the stump were followed until they deliquesced. Their lumina were small but free from clot.

POUPON.—*Embolie de l'artère fémorale dans la convalescence de la diphthérie* (*Progress méd.*, 1883, p. 10).

F., aged 7½ months, Feb. 17th, under care of Dr. Bouchut for "croup." Tracheotomy on the same day by M. Launois. She was discharged March 2d, with wound completely closed, and in very satisfactory health. During her first stay in hospital her urine showed some albumin, but there was none subsequently.

Re-admitted March 13th with intense dyspnœa, fever, and facial pallor. She was said to have discharged some false membrane in the interval, but



expectoration of masses of mucus are often mistaken for such by parents. There was no diphtheritic paralysis: temperature 39° C. (102° F.), respiration 40 per minute. Dulness at base of right lung. At this level there was a slight expiratory murmur with pleuritic rub. M. Bouchut diagnosed pulmonary embolism, thinking that the pleuritic effusion was of the nature of those effusions observed in the course of pulmonary embolism (Charcot). Auscultation of heart was not performed thoroughly on account of the respiration causing a considerable murmur over the tracheal wound, which had re-opened. Some mucous râles disseminated over thorax. Pulse 80.

March 14th patient complained of pain over the right popliteal space. In the evening there were violet-colored patches over the whole foot; they were the size of a franc-piece, and the violet hue was that of gangrene. The leg was cold and the pulsations in the popliteal and even in the crural artery were imperceptible. Temperature was 31° C. (88° F.) in right leg and 37° (98.6° F.) in the left. Sensation was absolutely abolished at the level of the patches of gangrene. Next morning (March 15th) Dr. Bouchut diagnosed embolism of the popliteal artery. The child died an hour after his visit.

Autopsy permitted on leg only. In the femoral artery, starting from the ring of the great adductor, and proceeding to the centre of the popliteal, a clot 3 centimetres (1.2 inches) long was found, looking like a pencil. The clot was brownish-gray in color and was not adherent to the wall. Here and there it showed clearer strata. It occupied the whole lumen of the artery.

Remarks: It seems clear that the clot in the femoral artery was due to an embolus around which the thrombus had formed, composing, as in all cases of this nature, almost the whole of the clot. This is a unique case in the history of diphtheria.

HYDE AND POWELL.—*Case of Raynaud's disease following diphtheria* (*Brit. Med. Journ.*, 1886 i, p. 203). (Reported by Allman Powell, M. B., House Surgeon.)

C. P., aged 48, admitted to Worcester General Infirmary, July 14, 1884. *History*: in Royal House Artillery, 22 years; drill-instructor to police 6 years. No illness except one or two "colds" from wet feet when on night duty. No syphilis, gout, or rheumatism. Family history good. On May 12th reported himself ill with sore throat. A week later had sudden loss of voice for which he was treated by a practitioner who said he had diphtheria. During convalescence his nose became blue and swollen in one night, resembling "an overgrown mulberry." This was followed in a day or two by similar appearances in pulps of fingers of right hand and in ears. In the following week the ring and little finger of the left hand were similarly attacked, causing pain. In three weeks the fingers were black along their entire length. The blackness then receded to the middle joints where blebs formed between the sound flesh and mortified parts. Subsequently black patches formed on the second and third toes of the right foot and along its outer margin. His mouth and tongue became swollen and painful, the latter being livid and an ulcer forming near the lip. His nose was obstructed but was cleared by his sneezing out some tough brown matter.

Examination, day after admission: Well built, fairly healthy-looking; has lost some flesh and become weaker from illness. Hair gray; no arcus senilis nor signs of general anæmia. Nose cold and livid. Edges of helices

both dark blue. Ungual, and part of middle phalanges of ring finger and little finger of right hand were black, dried up, and completely mummified. Remaining fingers of left hand and both thumbs were cold, glossy, devoid of hairs, and of a dusky red color at the tips. Unequal phalanges of second and third toes of right foot were also sphacelated. Sphacelated portions of fingers were separated from sound flesh by bullæ containing turbid serum. Physical examination showed chest normal. Radial, ulnar, and posterior tibial arteries normal. No paralysis, sensory or motor. Voice regained. Temperature normal. Urine contained little albumin, no casts or sugar. Blood showed increase of white corpuscles. Digestive functions good. Slight pain and tingling in fingers occasionally.

Improved rapidly under dietetic and tonic treatment. Went for change of air on Sept. 3. Gangrenous fingers amputated by Mr. Hyde on his return. Sloughs separated from ears on his return. Sloughs separated from ears and toes without interference leaving granulating surfaces beneath which healed rapidly. Stumps of gangrenous fingers slow in healing: thumbs and ears still cold and discolored, but he was well enough to resume work.

BRETON, M.—*Pralysie meta-diphtherique* (*La médecine moderne*, Paris, 1902, xiii, p. 216) (Société de Pédiatrie, Séance de 17 Juin, 1902).

M. Breton has had under his observation a little girl aged 3 years, suffering from hemiplegia due to multiple embolism, the sequela of diphtheria. The child suddenly manifested paralysis of the right side, and at the same time livid patches, followed by gangrene, appeared on the left leg. The hemiplegia persisted for eight months, although the child had the power of walking—dragging the leg along. There had been present in this case without doubt, endarditis, cardiac thrombosis, and multiple embolisms.

VEDDER, JOHN.—*Gangrene of the leg, caused by embolism following diphtheria* (*Trans. Med. Soc. State of New York*, 1879, p. 259).

Boy, aged 8 years, health had been generally good. First visited Dec. 21, 1877. Diphtheria, severe attack, but prognosis favorable. Throat and nares covered with thick membranous deposit; profuse discharge from nose; breath offensive; slight constitutional disturbance; fever moderate. Was visited every day up to Dec. 29. Sister taken ill with the disease meanwhile. Continued to visit her after convalescence was established in boy. On Monday, December 31st, nothing unusual in boy's condition, convalescence progressing well. In afternoon of same day he complained of numbness and pain in right leg from knee downwards, pain mostly in knee, below that, sensation of numbness. Leg cold and of a waxy appearance. Knee only painful, and upon slightest motion. Pain could not be produced in leg either by pinching or pricking with needle; leg dead from knee downward. It was white and cold. Next day, Tuesday, it was of a light mahogany color, which gradually grew darker till it was nearly black. Bottom of foot became quite dark, dry, hard, and shrivelled up in a few days. Pulse slow and weak at right wrist, normal at left. Respiration good. Mind clear. Jan. 5th or 6th, arm on right side paralyzed. Jan. 6th and 7th, slight paralysis of tongue and loss of speech. Died Friday, Jan. 11th, 2 A.M. Conscious during whole illness till a few hours before death. Two days before, he had complained of pain in left leg when it was moved. Patient of excitable temperament. No autopsy.

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## CHOLERA NOSTRAS—CHOLERA ASIATICA

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CHOLERA is, in the majority of cases, a water-borne disease, due to water having become contaminated with cholera organisms derived from some person suffering from the disease. It is, therefore, usually conveyed by drinking of water which has become polluted by the excreta or discharges of an infected person, as such water may occur in the form of ice, or may be found added to milk, or used for the washing of vegetables, etc. Ice, raw milk, uncooked vegetables, salads, and fruit may also transmit infection. Flies also, and other insects, especially ants, may be to blame by carrying the infection from polluted matter to food and drink, while these may be contaminated by the infected and dirty hands of those engaged in their preparation.

While in Hamburg, Germany, where I was appointed by the State as Cholera Physician in 1892, during the months of September, October, and November, we found out that even the money which is handled by so many people day after day, especially the pennies and dimes (*Pfennige, Kupfermünzen*) were carrying the Cholera Bacillus. Strong acetic acid was used for disinfection of the money. As everybody can remember, there was a big fight between the bacteriologists of three countries—Germany, Austro-Hungary, and Italy. In the middle of September the cholera had spread over these three countries, and especially Hamburg, Budapest, Genoa, and Florence were the headquarters of the Asiatic disease. There was no cholera in Russia. By that time Professors Scheuthauer, Babes, Weichselbaum, Petrik, Hoegyes, etc., had different ideas, Scheuthauer was against the washing and sprinkling

of the streets, while Babes and Hoegyes favored it. The victory was on Scheuthauer's side, as soon as they ceased to washing and let the streets dry for a few days, the cholera virulence ceased, the mortality sank from 80 per cent. to 50 and even 40 per cent. during its greatest virulence.

Ever since the Scheuthauer idea has been found the right one: All water intended for personal use, viz., drinking, cooking, washing, and, wherever possible, bathing must be boiled. Drinking water is best boiled in a can provided with a cover and a tap. The boiled and cooled water is then to be poured directly into the cup or tumbler. Care must be taken that an infected drinking vessel is not used, as, after the boiled water has cooled, it can be re-infected. Especially in the hot zone water from zeers and goulahs should be looked upon with suspicion unless these are carefully watched and cleaned. All raw milk must be boiled. Uncooked vegetables, raw salads, and fresh fruits, especially melons, should be avoided. Food stuffs should be carefully protected from flies, ants, and other insects and not stored anywhere in proximity to latrines or any collection of refuse. As far as possible all food should be cooked. Jellies are liable to become contaminated, and should be avoided. Care should be taken to see that cooks and other servants are cleanly in their habits and clothing. All dishes should be cleaned with boiling water and kitchen cloths should be well washed and dried in the sun. Any cloths used for straining soups, sauces, and the like should be washed in permanganate solution. Brooms, brushes, or cloths used for cleaning out latrines must on no account be used in kitchens or cook-houses. The *Spirillum*, found in "rice-water" fæces and intestinal contents, is motile, with end flagella, an aërobe, and facultative anaërobe, in short arcs and spirals, "comma" forms or longer winding filaments, 0.5 by 0.8 to 1.5  $\mu$  (1/50000 by 1/30000 to 1/15000 inch), and with no spores. It liquefies serum and gelatin; on plates forms white or yellow-white colonies with a granular surface and an irregular margin; in a gelatin stab liquefaction begins at the surface and proceeds downward, making an inverted cone with an air bubble at the top; in broth and liquid media it forms indol and nitrates, giving "cholera-red" reaction with pure hydrochloric or sulphuric acid ( $H_2SO_4$ ); milk

is not coagulated. Temperature of growth: 14°–42° C. (57° to 108° F.); best: 37° C. (98.6° F.). A spirillum resembling that of cholera asiatica is that of Finkler and Prior, obtained from fæces in cholera morbus; like the former in shape, growth, and indol formation, but longer, more slender, and coagulating milk; in gelatin stab it grows faster and liquefies faster; it is motile, with an end flagellum. Another is the spirillum of Metschnikoff, found in fæces of fowl cholera, shorter and thicker than that of cholera asiatica, but the cultures are very similar; not pathogenic for man; kills chickens, pigeons, and guinea-pigs. *Spirillum sputigenum*, isolated from saliva, resembles cholera, but is longer and thinner; it has not yet been cultivated in any medium.

Post-mortem examination, after death by asphyxia, demonstrates on the outer or serous surface of the intestines a peculiar mucilaginous film, perhaps more evident to touch than sight. The mucous surface is bright red or bluish in color with many small submucous hemorrhages. The contents of the intestine may be copious, thin, and watery, and contain small bits of desquamated epithelium, making the so-called rice-water discharge, characteristic of the disease. Blood may be mixed with this fluid, rarely a coloring of bile. The rice-water fluid is alkaline, has a peculiar stale odor, and contains sodium chloride, ammonium carbonate, and but little albumin. (Total solid matter from 1 to 2 per cent.) The mucous membrane is specially swollen in the lymphoid follicles, both solitary and agminated, when death occurs early (fourth day). The colon may be slightly hyperæmic and œdematous, and filled with rice-water fluid from the small intestine; at times early in the disease a pseudo-membrane forms, and after the algid stage a secondary fibrinous enteritis occurs in both small and large intestine. The microscopic picture is that of a coagulation necrosis and desquamation of the epithelium, confined to the tips of the villi or occupying the entire thickness of the mucosa. In the later stages of the disease the intestine may show but little of the rice-water material, bile is usually present, Peyer's patches are pigmented and diphtheritic membranes are found, especially in the lower part of the ileum.

To use Ziegler's own description of *Cholera epidemica seu asiatica*, I quote the following lines:

*Epidemic or Asiatic cholera* is characterized anatomically by the presence of an acute inflammation extending over the whole of the intestine, accompanied by an enormously copious transudation of liquid through the mucous membrane. In cases which prove fatal in the first two or three days, the bowel is found to contain a great quantity of a turbid, greyish, inodorous alkaline liquid, often mingled with minute shreds and flakes of mucus—the so-called “rice-water” stools. The mucous membrane is moist, pink, injected, and swollen, and here and there beset with hemorrhagic spots. Usually the serous surface is injected and turbid, and feels sticky to the touch. The epithelium of the more superficial portions of the crypts within the first few hours of the disease undergoes mucoid degeneration and desquamation, and the surface of the intestine becomes thereby covered with slime. Later on most of the epithelium exfoliates and mingles with the transuded liquid. The connective tissue of the mucosa, and to some extent of the submucosa also, is more or less thickly infiltrated with leucocytes, which sometimes penetrate even to the serous coat. The follicles of the small intestine are somewhat swollen, greyish-white or bright pink in color, and surrounded by a hyperæmic areola. The ileum is usually the part most affected, the large intestine being often almost unaltered, at least in the early stages.

When death does not occur until a later stage of the disease, the appearance of the intestine is notably different. The contents are scanty and less liquid, and at the same time they show more signs of the presence of bile: in the large intestine scybalous masses are sometimes found. The mucous membrane is pale or slate-colored, or it may be injected and beset with minute hemorrhages. Ulcers make their appearance in the stage of collapse, especially in the colon and lower part of the ileum, as the result of diphtheritic necrosis and sloughing. Sometimes the large intestine has almost the same look as in dysentery.

About the Cholera Bacillus says Robert Koch, the Great Discoverer, himself:

“Bei frischen Fällen finden sich grosse Mengen von Cholera Bacillen im Darminhalt und in den verschleimenden und sich abstossenden Lagen des Epithels, als auch in dem Lumen der Krypten und zwischen und unter deren Epithelzellen und im subepithelialen Bindegewebe. Die Spirillen bilden bei ihrer Vermehrung ein Gift, welches das Darmepithel schädigt und resorbiert, auf den Gesamtorganismus giftig wirkt, vornehmlich aber die Gefässe lähmt. Der saure Magensaft des gesunden Magens ist der Spirillen entwicklung hinderlich, Störung der Magenfunction prädisponiert danach zu Cholera infection.”

*Cholera nostras* appears very often sporadically only. The clinical symptoms also are not of pronounced character, and we see very often that it disappears very quickly. Nearly every autumn we have had cholera nostras in my country town, Teplitz-Schönhausen, where I was practising medicine. Especially about the middle of September the “little” epidemic, as we called it, began, and, as I can remember, there have been about six hundred cases nearly every year, in a town and farms of thirty thousand popula-

tion (mortality about 2 per cent.). But in the years of 1873 and 1892 we had about three thousand cases with a mortality of 25 to 30 per cent. Of course it was not so vehement as the cholera of 1873. German people call this epidemic "Die grosse Cholera"—the great cholera. About two hundred thousand cases had occurred by that time in the Empires of Central Europe. In Russia they had nearly one million cases, with a mortality of 75 to 80 per cent. In Germany and Austria the mortality average was 25 to 35 per cent. During the cholera in Hamburg people died very quickly, very often in a few minutes, but 2 or 3 hours was the common "term" set for the *exitus letalis*. There were sudden deaths in the street cars, omnibusses, everywhere. Whole families died out in a few days. I know of three families, where, out of nearly twenty people, fifteen members had met awful death by the cholera.

The general appearance of the cadaver is distinguished by tonic contraction of the flexor muscles, the arms and legs are drawn up and the muscles hard, the hands partly clenched, the face sunken, the nose and chin prominent, the surface generally cyanotic or only at the extremities. Hyperæmia of the pia mater, lobular pneumonia, hyperæmia and cloudy swelling in the kidneys, and shrunken liver and spleen are the common associated conditions. The vibrio may be obtained in pure culture from the intestinal mucus and the rice-water contents, and if death has occurred early sometimes from the gall-bladder, in later stages it may be impossible to find it (seldom after the twelfth day).

From a paper by O'Gorman one picks out the following practical points as likely to be of service. Note the stage when called to a case, as the treatment varies with the stages of the illness. Look for a blood-shot condition in the eyes, sometimes the only outward indication of reaction. Remember the differential diagnosis from arsenical poisoning. It is very well recommended at any stage to use calomel and sodium bicarbonate, repeated if rejected, until retained. The calomel increases the flow of bile, acts as an intestinal antiseptic, is sedative to vomiting, especially in frequently repeated fractional doses every quarter or half hour, is diuretic, antiphlogistic, and, taken continuously in doses short of toxic, stimulates the faculties, physical and mental. Soda aids its action, prevents sali-



vation and supplies vital element to the blood. In the first stage it is also advisable to administer intestinal antiseptics such as sulpho-carbonate of zinc, copper arsenite, peroxides, etc. For the rest, stop food, give fluids and try to prevent collapse. Carminatives, sedatives and astringents are useful and should be given. Chlorodyne, camphor, and opium among the drugs, and nuclein may prove very valuable owing to the increase of polynuclears it produces and its stimulation of cell growth. In the stage of collapse he utters a warning against alcohol, and states that there are only two great remedies, namely: atropine and strychnine. In the stage of reaction the occurrence of urination is a favorable sign and where there is danger of uræmia, pilocarpine may be tried. It is, however, risky and diaphoretics and hot coffee are safer and often efficient.

Choksy has a paper advocating cyanide of mercury in doses of 6 mgm. (1/10 grain) every two or three hours as a germicidal agent. He reports favorably on its use, the only drawback being a tendency to stomatitis during convalescence. In other directions his treatment is like that of O'Gorman. In acute delirium during the reaction stage bromide and hyoscyamus are indicated. McCombie reports very favorably from employing subcutaneous injections of hot normal salt solution, repeated whenever the pulse tends to fail. The addition of adrenalin chloride (1:1000) to the salt solution also seemed beneficial.

Rogers and Mackelvie speak highly of the value of large quantities of hypertonic salt solutions in transfusion for cholera. The strength they employ is about 1½ per cent. and they inject, as a rule, 2 L. (four pints) at a time, intravenously. Subcutaneous injections are only of benefit in mild cases. By this new procedure they believe the mortality has been halved. Other points of interest are the *latent cholera carriers* found by Gotschlich, at Tor, who, though they harbored true cholera vibrios, did not give rise to an epidemic and did not die of cholera, but from dysentery and gangrene of the bowel; and the quick agglutination method of diagnosis introduced by Dunbar and said to be reliable. It is as follows: Mix a particle of fecal mucus with one drop of peptone water and one drop of an 1:500 dilution of cholera serum (mixture a). Mix a similar particle with one drop of peptone water and one drop of 1:50 dilution of normal rabbit's serum (mixture b). Place each

mixture on a cover glass and examine as a hanging drop preparation. Observe agglutination in *a*, none in *b*; maximum result after about three hours. In this connection one must cite the work of Ruffer (*The Bacterial Diagnosis of Cholera*) which leads him to state that "it is not advisable to trust to the agglutination test only in bacteriological diagnosis of cholera. The test is useful, but not specific." It would seem then that the hæmolysis test must always be applied, for Ruffer noted no vibrio hæmolysis, when the agglutination test, Pfeiffer's reaction, and the fixation test were positive, while he states distinctly that "the agglutination, saturation, and Pfeiffer's tests are not in themselves of absolute diagnostic value for cholera vibrios. It is very important that these conclusions should be definitely confirmed or confuted, as they upset prevailing ideas on the bacteriological diagnosis of cholera.

## CONGENITAL SYPHILIS SIMULATING LEUKÆMIA AND SPLENIC ANÆMIA (BANTI'S DISEASE)

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THAT the clinical and hæmatological pictures of pernicious anæmia may be presented by cases of syphilis, usually of the late acquired form, is well known. Nearly every clinician having a large experience with cases of anæmia has seen cases of the pernicious type improve remarkably, or apparently become well, under antisypilitic treatment. Müller described four cases of anæmia in syphilis simulating the pernicious type, in one case the red blood-cells falling to 720,000. Laache and others have reported similar cases. In this form of syphilitic anæmia the blood-picture may in every respect, low count, relative high color-index, relative lymphocytosis, etc., be that of a typical pernicious anæmia.

The simulation of leukæmia by congenital syphilis is an occurrence, however, that has been overlooked by practically all the writers of text-books on hæmatology. It is surprising how meagre the statements are in such works concerning the condition of the leucocytes in syphilis, both the acquired and congenital forms. Cabot in his article on "Leukæmia" in vol. iv of Osler's "Modern Medicine" does not mention the possibility of congenital syphilis simulating leukæmia, although Osler and Churchman in vol. iii of the same system, in the article on syphilis, make the following statement on page 478: "And lastly, in certain cases of syphilis with enlargement of the liver and spleen the degree of leucocytosis is such that leukæmia is suspected. In a case of congenital syphilis with an extraordinary fissured liver, the spleen weighed more than 1500 grammes ( $3\frac{1}{2}$  av. lbs.). It was the most prominent feature in the distended abdomen. There was great increase of the leucocytes and the case was regarded as one of leukæmia until the post-mortem showed the picture of congenital syphilis." And again on page 481 of the same volume, "under the section on syphilis of the spleen the fact is noted that in certain of these cases there

may be a marked leucocytosis and a clinical picture resembling leukæmia."

That such a simulation by congenital syphilis is possible must be borne in mind in the differential diagnosis of leukæmia. During the last three years the writer has seen three cases of congenital syphilis in which the blood-picture was that of acute lymphatic leukæmia, but the autopsy findings were typically those of congenital syphilis with the characteristic changes in liver, spleen, and lungs, and the presence of spirochætes without any lesions typical of leukæmia of the known types. One of those cases was so striking that it is worth describing somewhat at length.

Female child, four weeks old, began to have hemorrhages from mucous membranes when four days old. Ecchymoses would appear spontaneously over the body and then gradually fade. Vomited blood during the first week after birth and passed blood in stools. In the fourth week developed an ecchymosis of left eyelid with the formation of a hæmatoma in the orbit causing marked exophthalmos. Skin was extremely pale and waxy; no trace of icterus, spleen and liver greatly enlarged. Flood showed about 2,000,000 reds, hæmoglobin 30 per cent.; leucocytes 40,000 to 60,000, mostly mononuclear, of large lymphocyte type. Great numbers of nucleated reds, normoblasts and megaloblasts were present. The atypical character of the white cells, these being mostly of the large bone-marrow lymphocyte type, with the great enlargement of spleen and liver and hæmophilia turned the clinical diagnosis towards hæmophilia due to leukæmia, but no positive diagnosis was given. Congenital syphilis was not included in the clinical diagnosis. Death took place from gradually increasing weakness, and the immediate cause of death was supposed to be the anæmia resulting from some internal hemorrhage.

*Autopsy Protocol.*—Female child, weighing 2500 Gm. ( $5\frac{1}{2}$  av. lbs.), and measuring 48 cm. (19 inches). Intertrochanter measurement 9 cm. ( $3\frac{1}{2}$  inches); shoulder-breadth 12 cm. ( $4\frac{3}{4}$  inches); circumference of head 35 cm. (14 inches); length of finger-nails 5 mm. ( $1/5$  inch); length of hair 12 mm. ( $1/2$  inch); anterior fontanelle 2.75 cm. (1.1 inches). -Left eye shows marked exophthalmos, mass size of small orange filling left orbit; eyelids ecchymotic. Pressure upon orbital mass causes spurting of blood. Orbital tissue suffused with blood. Skin extremely pale, waxy, no ecchymosis visible, no jaundice, scleræ clear; no hypostasis; no œdema. Very little panniculus; umbilicus negative; skull negative. Brain extremely anæmic, very soft and jelly-like. Thymus large, very pale, weight 4 Gm. (1 dr.), extends short distance over heart. Pericardial fluid slightly increased, clear amber yellow. Heart enlarged, dilated; weight 40 Gm. (1.3 troy ounces); length 4.5 cm. ( $1\frac{4}{5}$  inches); nearly round; very pale in color, almost cream color; auricles and ventricles moderately dilated. Ventricle walls greatly thickened 8-10 mm. (0.3-0.4 inch); right ventricle wall thicker than left. Muscle extremely pale, but moderately firm. Numerous subepicardial ecchymoses the size of a pin head.

Foramen ovale patent, likewise the ductus arteriosus. Valves negative. Pulmonary arteries negative. Numerous small yellowish spots in intima of aorta.

Lungs voluminous and extremely pale; mottled with dark-red, firm, airless areas. Consistency of lung increased. Pale portions air-containing; darker areas airless or nearly so. On section the latter show increase of stroma and obliteration of alveoli resembling early stage of white pneumonia. Left lung weighed 22 Gm. ( $5\frac{3}{4}$  drachms), right one 30 Gm. ( $7\frac{3}{4}$  drachms). Pleuræ negative; no fluid in pleural cavities. Mouth and neck organs negative, with exception of lymph-nodes, all of which are enlarged, pale, and firm. Bronchial and posterior mediastinal nodes enlarged and pale. Homogeneous on section.

No fluid in peritoneal cavity. Spleen as large as that of an adult, weight 140 Gm. ( $4\frac{1}{2}$  ounces), capsule stretched; consistence firm. Surface mottled near upper pole. On section very anæmic, pulp firm, stroma increased, follicles less evident than in normal spleen.

Adrenals large, very pale and firm, medulla dark red, diminished in amount. Kidneys, right weighed 20 Gm. ( $5\frac{1}{4}$  drachms), measured  $5.5 \times 3 \times 2.5$  cm. ( $2.2 \times 1.8 \times 1$  inch); left weighed 22 Gm. ( $5\frac{3}{4}$  drachms), measured  $5 \times 3 \times 2$  cm. ( $2 \times 1.8 \times 0.8$  inch). Fatty capsule fairly well developed. Fetal lobulations preserved. Color of both kidneys very pale; otherwise no changes. No uric acid infarction. Gastro-intestinal tract extremely pale; no blood, or traces of recent hemorrhage found. Mesenteric nodes large, firm, very pale. Pancreas very pale and firm. Liver enlarged, extended one hand-breadth below level of ribs in median line, extended entirely over the stomach to meet the spleen; edges rounded, surface smooth, pale brownish in color, slightly mottled with darker brown; consistence firm. On section the surface showed a small amount of pale blood. Stroma increased, surface nearly homogeneous, vessels enlarged.

Centre of ossification in lower epiphysis of femur measured 2 mm. ( $1/12$  inch). Genital tract negative. Retroperitoneal lymph-nodes greatly enlarged, very pale, firm and homogeneous on section. Sternum sclerotic; practically no red marrow. Large bones showed very pale lymphoid marrow. Section of the left orbit showed the presence of a recent hæmatoma.

A blood-count made just before the autopsy showed about 80,000 white cells, 80-90 per cent. of the lymphocyte type, the majority being atypical cells of the bone-marrow type of the large lymphocyte variety while a small per cent. consisted of cells of plasma-cell type. This finding, in connection with the appearances of the liver and spleen when the abdomen was opened, made a diagnosis of acute lymphatic leukæmia the most likely thing to be considered. Smears of the blood taken at the autopsy presented a picture not to be interpreted otherwise than as the picture of blood of an acute lymphatic leukæmia. The orbital hæmatoma and subepicardial petechiæ, hypertrophy of heart, and very pale, firm enlargements of all organs, all supported a gross pathological diagnosis of leukæmia. Only in the case of the lung was a suspicion of congenital syphilis apparently justified by the gross appearances. A tentative diagnosis of acute lymphatic leukæmia with orbital hæmatoma was made.

The greater portion of each organ was preserved fresh in the cold room until the next morning, when smears were examined with the dark field microscope and found to contain an enormous number of *Spirochæta pallida*. Stained smears showed them as well.

The microscopical examination of all the organs revealed the changes char-

acteristic of congenital syphilis. The liver showed a marked picture of increase of reticulum throughout the lobules,—the atypical type of congenital syphilis,—with numerous collections of lymphoid cells, not only in the periportal connective tissue but also throughout the lobules, resembling the collections of deeply-staining lymphoid cells found in the fetal liver, but greatly increased in size. All the blood-vessels showed the marked lymphocytosis. The spleen showed a disappearance of the follicles and great increase of the stroma, changes characteristic of congenital syphilis. Levaditi preparations of both of these organs showed enormous numbers of *Spirochæta pallida* in the blood spaces and vessels and in the tissue spaces.

Typical areas of white pneumonia were found in the lungs, and these likewise contained enormous numbers of spirochætes. Sections of the heart-muscle showed diffuse proliferation of the stroma with localized areas of interstitial myocarditis richly infiltrated with cells of the lymphatic type. Many of the blood-vessels showed perivascular infiltrations and the subepicardial petechiæ corresponded to similar areas of proliferation of the stroma and infiltration with atypical lymphoid cells. Levaditi preparations of the heart showed these areas to contain enormous numbers of spirochætes.

Similar findings occurred in the lymph-nodes, pancreas and adrenals. The kidneys showed the least change. Hyaline glomeruli were found to the number of 6-8 in a small section. The blood-vessels of all organs showed a condition of lymphæmia, but this was most marked in the vessels of the liver, spleen and lungs. The bone-marrow was fetal in type and showed no changes that could be interpreted as leukæmic. The condition of the lymph-nodes was, likewise, non-leukæmic in character.

The question may be raised if in these three cases the leukæmic condition of the blood may not have been due to the co-existence of a true leukæmia. The findings in the bone-marrow and lymph-nodes showed no changes that could be called the primary and essential lesions of leukæmia. Much more probable, it seems to me, is the view that the leukæmic blood-state is of *histogenous* origin and not *myelogenous*. The atypical character of the cells in the blood, their identity with the cells in the innumerable foci of infiltration found in all the organs, particularly the liver, heart, lungs, and spleen are the grounds upon which I base such a belief. The intensity of the spirochæte invasion, the organs and tissue being literally crowded with them, and the endothelial, lymphocyte, and fibroblastic reaction and proliferation make it quite possible for great numbers of young cells of the lymphocyte type (particularly the large atypical form) to gain entrance to the blood-stream and circulate there as leucocytes. I am inclined to class the third case, therefore, as one of *histogenous leukæmoid condition of the blood* due to

congenital syphilis. Such leukæmoid conditions of the blood might be very appropriately styled *pseudoleukæmias*, had not this term been appropriated to other usages.

*Simulation of Splenic Anæmia by Congenital Syphilis.*—I have seen the material of but one case of this kind, a girl, aged fifteen years, with symptoms diagnosed clinically as splenic anæmia, an enormous spleen filling in the left side of the abdomen, secondary anæmia, no leucocytosis, ascites, small nodular liver, general marasmus and non-development. These symptoms all pointed to splenic anæmia, and probably to beginning Banti's disease. Splenectomy was performed and a five-pound spleen removed. The patient died three weeks afterwards with symptoms of respiratory embarrassment. The left pleural cavity contained a large amount of fluid. The spleen showed a number of large caseous nodules. On microscopic examination a typical condition of syphilitic fibrosis with multiple gummata was found. Liver cirrhotic, containing numerous caseous nodules. On microscopic examination it showed a typical liver of congenital syphilis with multiple gummata. A pathological diagnosis of congenital syphilis was, therefore, made, and further inquiry into the clinical history confirmed the pathological diagnosis, if such confirmation could be thought necessary.

In conclusion, my experience agrees with the statement made by Osler and Churchman that congenital syphilis may simulate leukæmia. Indeed, I believe that an *atypical leukæmic condition of the blood of histogenous origin* may arise in congenital syphilis of intense infection and reaction, atypical cells of the large lymphocyte type entering the circulation from foci of infiltration and proliferation throughout the body, particularly in the liver, spleen, heart, and lungs. The simulation of splenic anæmia by congenital syphilis is more obvious. In the absence of history and other evidences of congenital syphilis a differential diagnosis may be impossible without recourse to the more specific methods of differentiating syphilis (Wassermann reaction, removal and examination of tonsils, lymph-nodes, etc., for spirochætes). At any rate the possibility of congenital syphilis in cases showing the splenic-anæmia complex must be borne in mind. Moreover, it must be borne in mind that the symptom-complex of Banti's disease, or splenic anæmia, may be the result of syphilitic disease of the portal vein, as in two cases reported by Borrmann (*Deut. Arch. f. klin. Med.*, 1897).

# Surgery

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## TRANSFUSION OF BLOOD EMPLOYING ONLY VEINS \*

BY GEORGE MORRIS DORRANCE, M.D.

AND

NATE GINSBURG, M.D.

PHILADELPHIA

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TRANSFUSION is not a recent treatment for hemorrhage, but dates back several centuries. The technic of performing transfusion has recently come to occupy a prominent place in surgical literature. It has from time to time been popularized or revived by communications treating of new instruments for its performance, or by case reports where resort to this operation was done as a last means to save a life that was slowly ebbing away as a result of uncontrollable hemorrhage. This means of supplying a deficiency in the quantity and quality of blood in a subject of grave hemorrhage is still too infrequently utilized. No doubt the absence of the tubes and a simple technic for anastomosis of blood-vessels will account in many cases for the failure to offer a bleeding subject what little opportunity exists for tiding over a critical period in the course of a serious disease or accident by performing transfusion. Even in the absence of tubes, metal or otherwise, direct suture of the vessels will suffice for the performance of transfusion, although this operation is more difficult to perform, necessitating direct end-to-end approximation of the vessels of the donor and of the recipient. Direct suture of the vessels is tedious and far more difficult than the use of metal cannulas. While in the hands of those familiar with blood-vessel surgery it may be easy of performance, it is to one first attempting transfusion difficult, tedious, and frequently impossible.

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\* From the Laboratories of Experimental Surgery in the University of Pennsylvania.



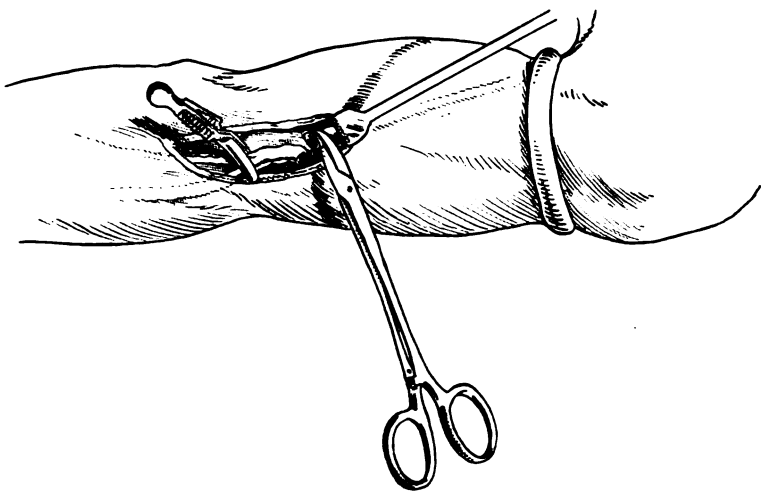
The authors have performed a number of experimental transfusions upon dogs in the laboratories of experimental surgery in the University of Pennsylvania, for the purpose of testing numerous instruments now in use, and if possible arriving at a suitable technic for all cases. Experience has taught workers in this field a lesson which we have emphasized in an earlier communication on this subject; that to popularize this operation for all cases demanding an immediate supply or to replace blood in a depleted vascular system, some procedure must be announced whose simplicity of performance will readily commend it to anyone desiring to resort to transfusion.

Transfusion of blood performed by the artery-to-vein method is both difficult and attended by some danger. We have, after a rather large experience in human transfusion, entirely discarded this method, because of the technical difficulties encountered, and on account of the occurrence of two cases of acute dilatation of the heart. A careful review of the literature on this subject is quite convincing of the fact that others have had this same unfortunate experience. This clinical picture occurring during the course of blood transfusion is not of infrequent occurrence in the experimental laboratories where dogs are employed.

The flow of blood from the vein of the donor is of sufficient pressure to readily flow into any of the veins exposed in the leg or arm of the recipient. The successful application of this method of vein-to-vein anastomosis for performing transfusion in a number of recent cases has been most gratifying. The exposure and juncture of the veins is a matter of only a few minutes, and that sufficient blood is transfused is only too well known to us, by the substitution in one of our cases of the rosy-red hue of the face of the donor for that of the recipient, and the subsequent occurrence of shock in the donor. Since blood flows with less rapidity in the veins, the duration of transfusion is necessarily longer; but this is only a question of secondary importance. The operation of vein-to-vein transfusion as advocated by us is simpler and easier of accomplishment than the artery-to-vein method, owing to the absence of technical difficulties attending the other methods.

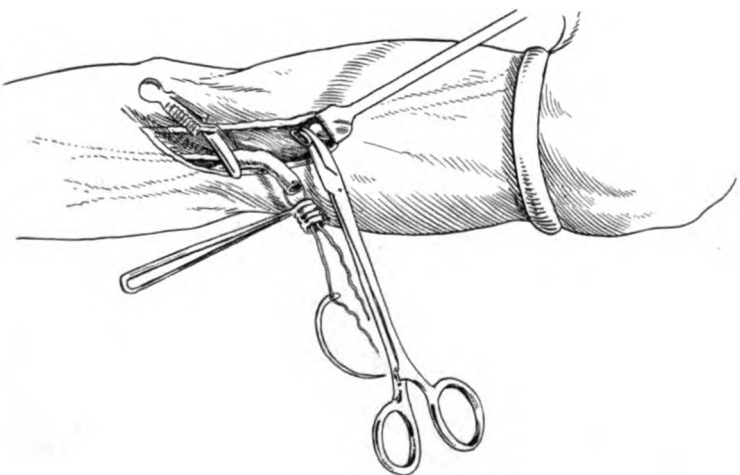
The vein-to-vein method of transfusion is simple and easily performed. The superficial veins are employed, and the danger of

FIG. 1.



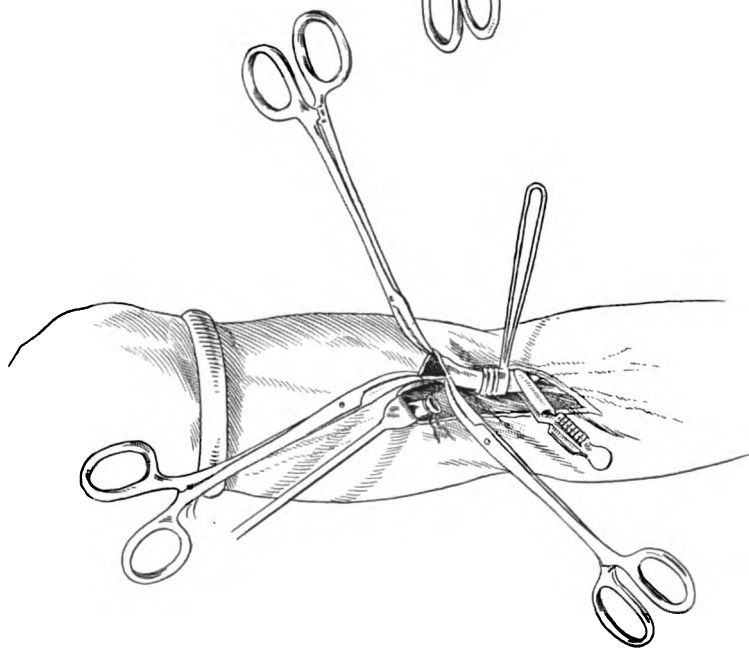
Exposure of the vein of the donor.

FIG. 2.



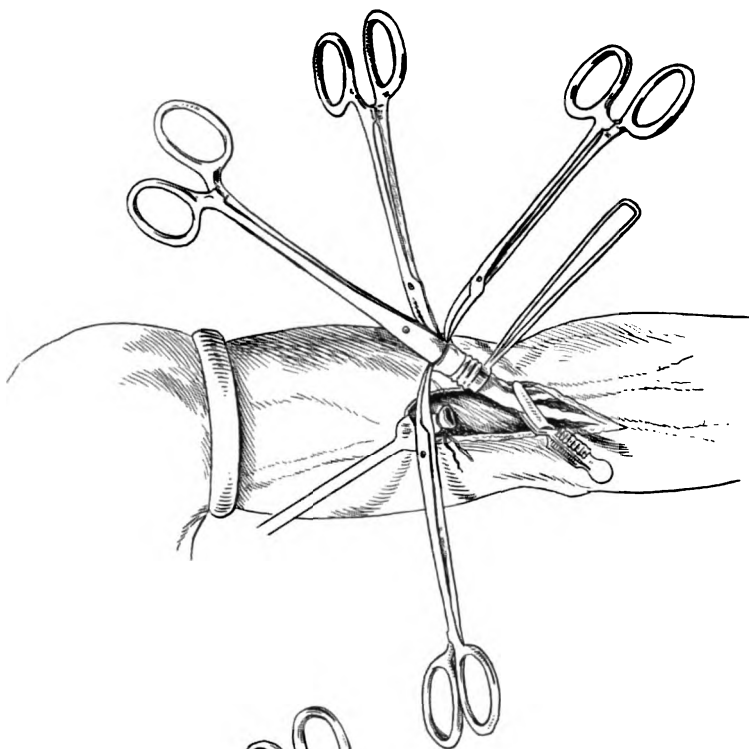
Threading the cannula upon the vein by means of a silk suture.

FIG. 3.



End of the vein grasped by three small hemostats equally distant apart.

FIG. 4.



A hemostat inserted into the vein to distend it and to assist in the retraction of the veins over the cannula.

acute dilatation of the heart is obviated by the slow-flowing current of blood. In a number of experiments in which this method was employed, no symptoms of acute dilatation occurred.

The question of the choice of a method of securing the juncture of the vessels has been determined by a careful series of experiments. The direct suture method was employed and found to give very satisfactory results; but we have discarded it, since its performance is too complicated and difficult ever to come into general use.

The method first advocated by Payr is easily performed, and satisfactory in every way, employing the modifications of Payr's original cannula by Crile and Sweet. The tubes of metal, glass, or other materials, lined with paraffin or vaseline, recently revived by Brewer and Fontleroy, were given a thorough test, but were discarded because of the early clotting which occurred. Clotting occurred usually within twelve minutes, and often as early as one minute from the time of the introduction of the glass tube.

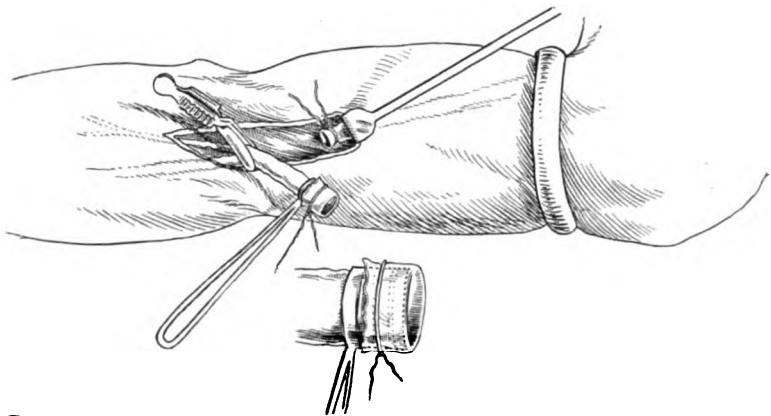
*Selection and Preparation of the Donor.*—The donor should, if possible, be one of the same family, moderately young, and free from any organic disease of the heart, etc. A careful history, and an examination for syphilis, should be made, and if time permits the Wassermann test for syphilis should be done. While a blood and a urinary examination are not essential both are advisable. The tests for hæmolysis are not very satisfactory, and are not of much value. This complication is certainly of rare occurrence, and when it does follow transfusion, is probably the result of obscure blood changes which were taking place before the transfusion was undertaken. The arm of the donor should be constricted just below the axilla by a tourniquet sufficiently tight to distend the superficial veins, but not to obstruct the arterial flow. In this way the largest vein with the fewest tributaries can be outlined and selected for use. The arm is then properly sterilized and the site of incision rendered anæsthetic by hypodermic injections of cocaine or eucaine, employing a 1 per cent. solution. It will usually be found that the median cephalic vein is the one selected for use.

The preparation of the recipient will consist in the dilatation of the veins and the sterilization of the arm, as narrated above.

*Technic of Vein-to-Vein Transfusion.*—An incision about three

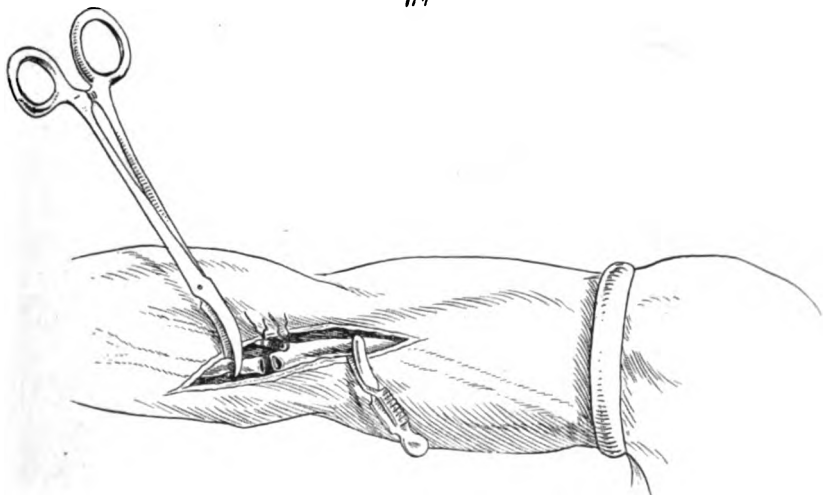
inches in length following the course of the vein will usually be sufficient. After exposure of the vein in the donor, a rubber-covered clamp is applied to the vessels at the lower angle of the wound, and the vein is grasped at the upper angle by a hæmostat. The vessel is next divided immediately below the hæmostatic forcep (Fig. 1). A small, round, pointed needle, threaded with fine silk, is passed through the end of the vein, to assist in threading the cannula upon the vein (Fig. 2). The upper proximal end of the vein in the grasp of the hæmostat is ligated and the hæmostat is removed. The open distal end of the vein which has been passed through the cannula is grasped by three mosquito hæmostats, applied equal distances apart (Fig. 3). When traction is made upon the hæmostats the lumen of the vein assumes a triangular shape. A hæmostat should now be introduced into the vein for the purpose of distending it. At the same time the cannula is pushed up against the hæmostat to prevent the vein from retracting (Fig. 4). The protruding end of the vein should be drawn down or everted over the cannula, forming a cuff, by gentle traction on the three hæmostats. A ligature is tied around the everted portion of the vein covering the cannula, and the hæmostats are removed (Fig. 5). The distal end of the vein of the donor is now prepared for insertion into the proximal end of the vein of the recipient. Exposure of both veins should be made simultaneously, and as soon as the cannula is fixed in the vein of the donor, the next step in the operation should be the juncture of the two veins. Since the blood current is to be directed toward the heart of the recipient, the exposed vein in this subject is divided and the distal end clamped and tied with a ligature. The proximal portion of the vein is compressed by a rubber covered clamp, and is then prepared for the insertion of the cannula previously applied to the vein of the donor (Fig. 6). The upper open end of the vein is then grasped by three mosquito hæmostats, equal distances apart, and the vein is distended, as was done in the case of the donor (Fig. 7). The arms of the recipient and donor are then placed side by side. This will bring the left arm of one into relation with the right of the other. The cannula carrying the vein of the donor is invaginated into the distended vein of the

Fig. 5.



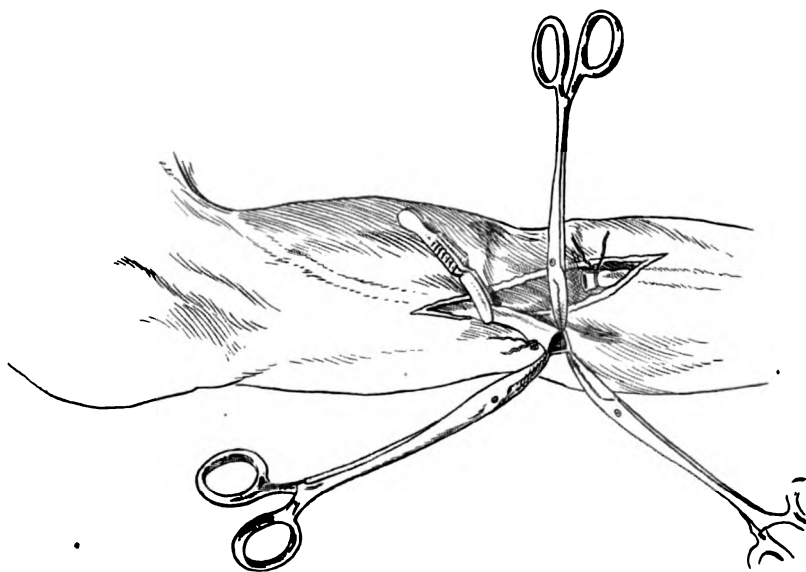
A cuff of the vein reflected over the cannula.

Fig. 6.



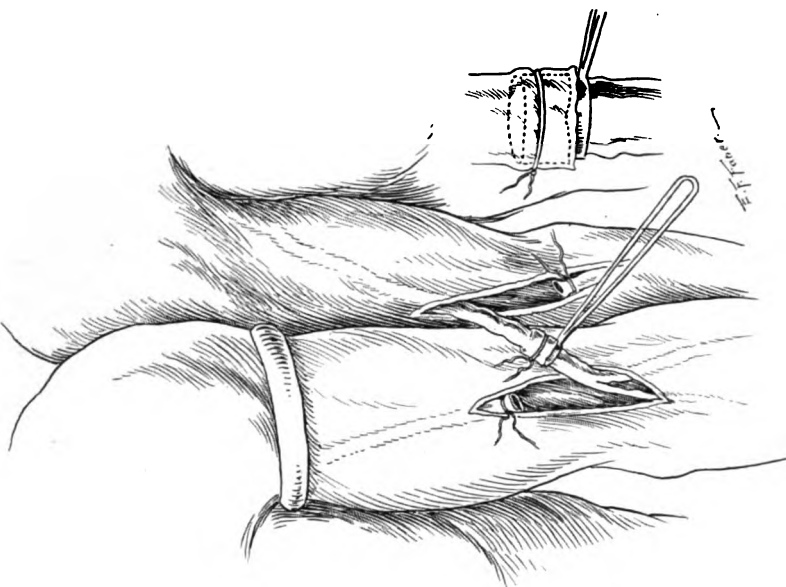
The exposure of the vein of the recipient.

FIG. 7.



The proximal end of the vein of the recipient grasped equally distant apart by three small haemostats.

FIG. 8.



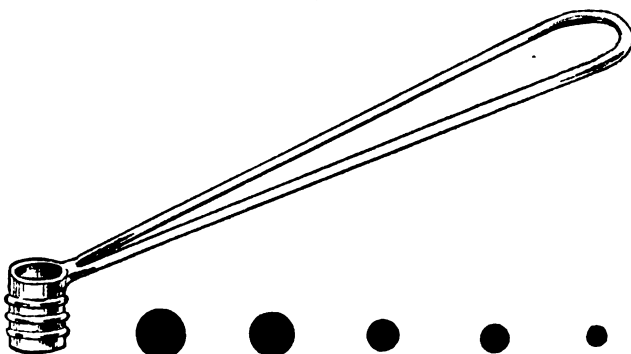
Insertion of the cannula with the reflected cuff of the vein of the donor into the distended end of the vein of the recipient.

recipient and is held there by a ligature. All forceps are released (Fig. 8).

The rubber covered clamps on the veins of the recipient and donor should be removed and the blood allowed to flow. The tourniquet should be removed from the arm of the recipient, and that on the arm of the donor should be slightly loosened. If the veins in the arm of the recipient are very small, it may be necessary to employ the internal saphenous vein of the thigh, as shown in Fig. 9. The cannula and sizes which we employ are shown in Fig. 10, the dotted figures indicating the variations in the lumen.

The indications for performing transfusion should include the following diseases or conditions where severe hemorrhage of sudden onset threatens the life of the patient. Gastric ulcer, typhoid fever,

FIG. 10.



Sweet's cannula. The round black figures represent the different sizes.

and certain blood diseases, as hæmophilia and hemorrhage of the new-born. Severe uterine hemorrhage, whether post-partum or incident to some factor impairing the integrity of the mucous lining of this organ, will often call for a rapid performance of this operation.

The virtue of transfusion of blood lies in the fact that the corpuscular content of the blood is increased, and in many cases the coagulation index is increased, thereby stopping bleeding.

Messrs. Burleigh, Hayward, Pitt and McElhone, undergraduates in the University of Pennsylvania, have assisted in the experiments.

A report of the last three of our 18 cases will be sufficient to show the results thus far obtained.

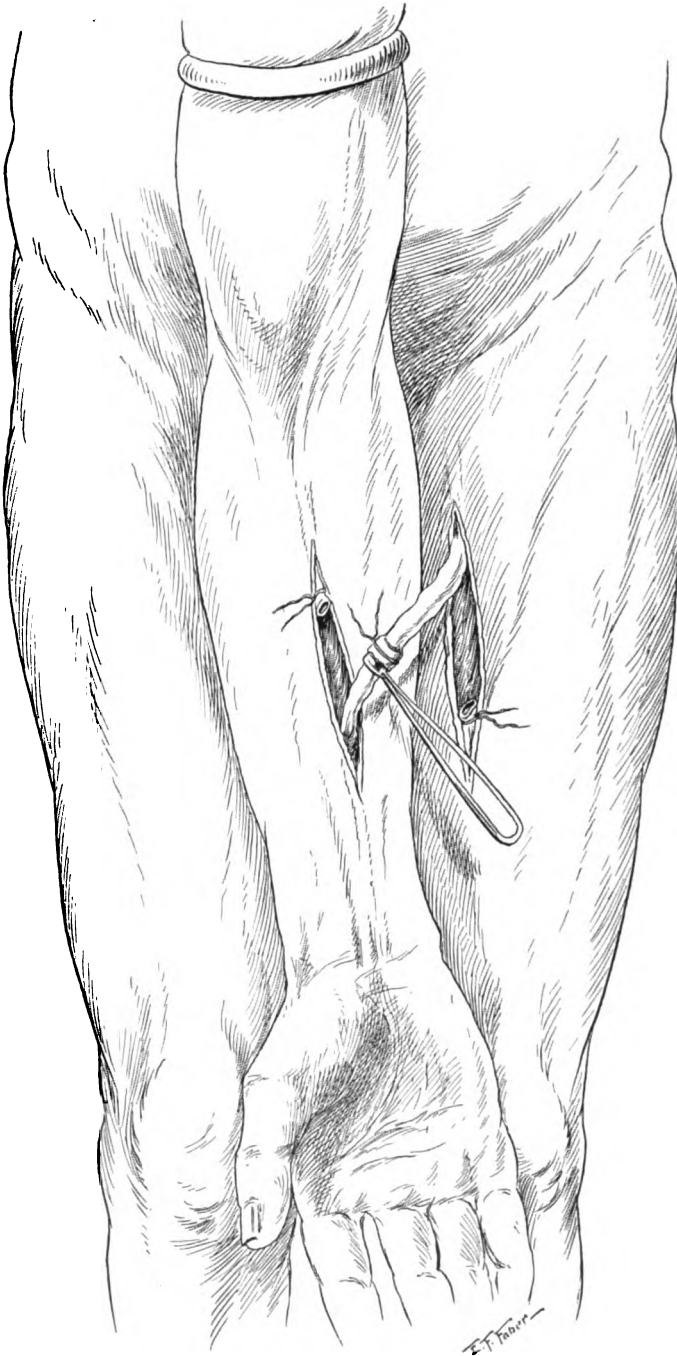


CASE XVI.—Mrs. B. was seen in consultation with Drs. W. P. and E. M. Kistler, of Allentown, Pa., who assisted at an immediate abdominal section for ruptured extra-uterine pregnancy with very severe bleeding. After the operation the patient was almost pulseless, and vein-to-vein transfusion from husband was performed, an immediate return of the volume and a marked decrease in the rapidity of the pulse taking place, and there was a disappearance of shock and pallor. An uninterrupted recovery followed.

CASE XVII.—Mrs. H., had suffered with continuous bleeding for the past eight months from an intra-uterine fibroid. Before transfusion the patient was quite pulseless, with the lips and conjunctiva almost free from blood. The patient being unable to stand an operation, vein-to-vein transfusion from son was practised. Thereupon some difficulty was encountered on account of the small size of the veins in the arm of the recipient. The operation for the removal of the fibroid was performed the following day under ether anæsthesia. An uninterrupted recovery took place. This patient was seen in consultation with Drs. W. P. and E. M. Kistler, of Allentown, Pa., who also assisted at the operations.

CASE XVIII.—Mrs. B. was operated at St. Agnes Hospital, the patient being seen in consultation with Drs. R. M. Goepp and Alfred Allman, of Philadelphia. Very severe gastric hemorrhage had almost exsanguinated the patient, the hæmoglobin being but 25 per cent. and the red corpuscles 1,500,000, while the white corpuscles were increased to 32,000. The pulse was rapid and thready. Vein-to-vein transfusion from a brother-in-law was performed. There was an immediate return of color and an increase in volume and quality of the pulse. During the transfusion the hæmoglobin gradually increased until it reached 65 per cent. An immediate exploratory section was performed under ether, the patient left the table in good condition but died on the following day from a continuation of the disease.

**FIG. 9.**



**Superficial veins of the forearm of the donor connected with the internal saphenous vein of the thigh of the recipient.**



## THE TECHNIC, AIMS, AND LIMITATIONS OF SPINAL ANÆSTHESIA IN THE YOUNG

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THE object of this communication is to attempt, in the first place, to supply deficiencies in previous publications on this subject, and, in the second place, to lay before those who wish to adopt spinal anæsthesia in children such practical bearings of the question, gleaned from a personal experience of between three and four hundred operations, as may assist them to continue this study from that point which the writer has reached at the present time. Owing largely to want of space, previous papers have dealt mainly with theoretical issues, and the influence of these on (1) the amelioration of certain disadvantages attendant on the employment of spinal anæsthesia, (2) the prevention of absorption of the toxic agent into the blood stream, and (3) the acquisition of a satisfactory control over the distribution and effects of the drug employed. A statistical analysis of cases was published in support of the views maintained; <sup>1</sup> repetition of these facts, therefore, is unnecessary, more detailed information being obtainable in those papers.

Briefly, it has been shown that the important factor of diffusion was responsible, to a large extent, for the distribution of the drug introduced in the spinal theca; that, the cranial sinuses being the chief seat for the absorption of salts contained in the cerebrospinal fluid, circulatory changes were induced which not only gave rise to faintness and pallor, but also favored the absorption of the active agent, and led to symptoms of poisoning: further, that diffusion also vitiated and masked, to a considerable degree, the possibility of accurate localization of the area of paralysis by the aid of gravity, and rendered efforts to induce a complete anæsthesia dangerous, owing to the liability of poisoning or the spread of the paralysis to vital structures.

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<sup>1</sup> *Lancet*, Sept. 25th and Oct. 2d, 1909; and June 11, 1910.

Stovaine was chosen, owing to its powerful action, and to the fact that it is very slowly diffusible and therefore less toxic. Diffusion was still further suppressed by the employment of dextrin as an indiffusible vehicle for the stovaine.

With the employment of dextrin-stovaine it has been shown possible to control the distribution of the drug by means of gravity with a considerable degree of accuracy; to keep the drug in the spinal theca, where absorption takes place to a very small extent only; and so to concentrate the action of the stovaine on a small area of the spinal nerve roots that a much stronger dose of this drug can be employed with safety, and a complete anæsthesia be obtained in the desired situation. The solution advocated at present (*Lancet*, June 11, 1910) is that which has yielded the best results and, therefore, this paper concerns spinal anæsthesia induced by fluids of a similar composition.

TECHNIC OF ADMINISTRATION.—1. *The Management of the Child*.—It is well known that, in surgical enterprises, insufficient attention to minor details is frequently responsible for failure; this is the excuse for laying some stress on the importance of the correct management of children as a factor of influence in the success of the method under consideration. It is advisable, when possible, that the nurse in charge of the case be known to the child; this will often be impossible, hence the nurse should not only be familiar with the routine of administration, in order that each step may be carried out quickly and quietly with the minimum of discomfort to the patient, but she should be experienced and sympathetic in the management of children. The influence of the psychical element on the successful termination of the operation is a consideration of some weight, and must not be neglected; and, if the nurse and the surgeon combine to do their respective duties quickly, quietly and without hesitation, the patient will not be alarmed, but the injection will have been given and the child turned over and made comfortable before he is well aware of what has occurred. Speed is essential. Even nervous children are, as a rule, quite easily managed; ignorance is their safeguard against panic, and I have only on one occasion encountered any difficulty in this respect; even then the progress of the operation was not impeded.

In the case of children who are very ill and in great pain,—

occasions when spinal anæsthesia is most likely to be called for,— it is encouraging to remember that the management of the patient presents no difficulties; any disturbance resulting from the skin or lumbar puncture is only temporary, for, so great is the relief from pain as the anæsthesia becomes complete that children quickly become quiet and often will sleep. It may be stated generally that the younger the patient, the simpler will be the management, infants being the most amenable to the method.

FIG. 1.



Position of the patient with the hands clasped beneath the thighs. The simplest way for the nurse to control the child in this position is seen. The surgeon's left little finger is on the crest of the ileum, the left forefinger is between the third and fourth lumbar spinous processes. Everything is ready for the lumbar puncture.

2. *Instruments Required.*—A hypodermic syringe, with aluminium or platino-iridium needle is used for anæsthetizing the skin. Steel needles should not be employed, as they quickly get rusted, or will break rather than bend if the child moves. For the lumbar puncture, the instruments recommended by Mr. Barker<sup>2</sup> cannot be improved upon. These should be either nickel plated or made of aluminium, since, the action of the stovaine being vitiated by the presence of an alkali in the water used for boiling ordinary instru-

<sup>2</sup> Brit. Med. Journ., Mar. 23, 1907; and Feb. 1st and Aug. 22, 1908.

ments, water alone must be used for sterilizing, and steel instruments would quickly rust.

3. *Steps of Administration.*—The child is placed on his right side, the head and knees well approximated so that the back is well rounded, and the hands clasped together beneath the thighs. The nurse talks to the child and maintains this position, which has the advantage that, if the patient starts when the puncture is made, the pull of the arms tends to prevent extension, which would add

FIG. 2.



Position in which to hold the patient for 15 or 20 seconds, directly after the injection, if a high anaesthesia (sixth or seventh dorsal segment) is required; X marks the lowest point of the curve of the back to which the fluid will gravitate.

to the difficulties of the manoeuvre. In this position the skin of the back is cleansed.

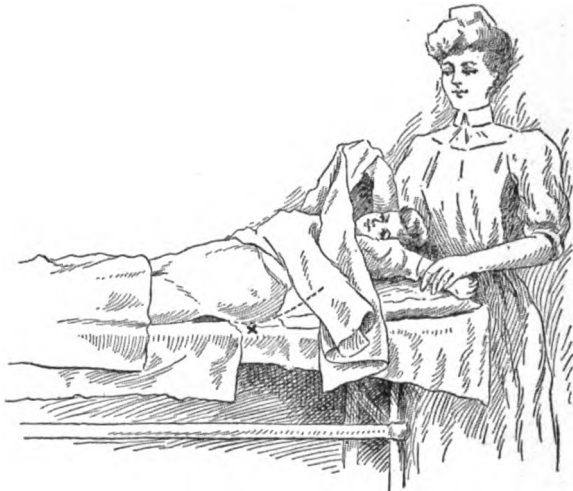
It is a good plan to prepare the operation area and place a sterile towel over it before the injection of the anaesthetic agent (when this step is possible), so that the full duration of anaesthesia may be at the disposal of the surgeon.

In this position (Fig. 1) about ten to twenty minims of a three per cent. solution of eucaine are introduced under the skin at the site of the intended lumbar puncture, and an area of about a quarter of an inch is thoroughly anaesthetized. It is this preliminary step which is most likely to disturb the patient, and,

therefore, the needle should be very fine and sharp, and the puncture made quickly and cleanly. The object of the preliminary anæsthetization of the skin is to render the lumbar puncture practically painless, so that the child will keep quite still during its performance, and speed and accuracy will be assured.

As regards the site of puncture, the second, third or fourth lumbar intervertebral spaces are available, the third being the one usually selected. I do not consider it necessary or justifiable to puncture above the second space, since, with the employment of a heavy fluid of a low diffusibility, the level of the paralysis can be

FIG. 3.



Ready for operation after manoeuvre shown in Fig. 2; X, lowest point of curve.

quite easily regulated by the position of the body wherever the puncture is made.

The technic of the *lumbar puncture* is simple and easy, and the principles involved are two-fold: (a) To puncture the dura in the mid-line, and (b) to select the simplest mode of access. Now, when the back is rounded, the site which offers the greatest space for the needle is between the laminae, immediately to one or other side of the spinous processes.

The index finger of the left hand is placed between the two spinous processes at the selected level, and the hollow needle containing its obturator, resting against the palm of the right hand and



directed by the right index finger, is entered at the anæsthetized spot with its shaft touching the left index finger, and is then pushed smartly in a direction slightly upward and toward the mid-line.

In its course the needle can be felt to penetrate two resistances: (1) The ligamentum subflavum, and (2) the dura mater. If the skin has been properly anæsthetized, children will frequently be unaware of the lumbar puncture, which may not even disturb them in a conversation. Two additional advantages may be cited in favor of lateral puncture: (a) Approach through the largest space diminishes the liability of touching periosteum, which is sensitive

FIG. 4.



Position in which to hold patient for anæsthesia to the umbilicus (tenth dorsal segment); X, lowest point of curve.

and may cause considerable disturbance. (b) If the back is suddenly straightened, the needle, by lying to one side of the spinous processes, is less likely to be bent or broken.

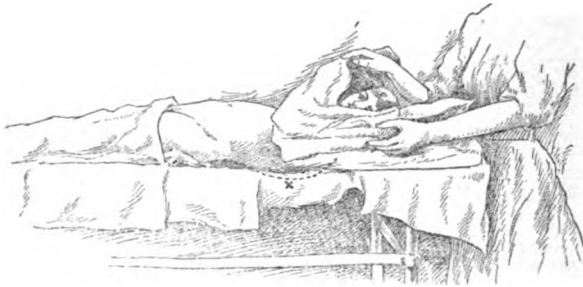
The stylette is then withdrawn from the needle, *which is now pulled out to the greatest distance compatible with a free flow of cerebrospinal fluid*; this is allowed to run out until its flow is obviously influenced by respiratory movements, for two reasons: (1) Absorption of stovaine into the circulation is still further delayed if the pressure in the craniovertebral cavity is lower than that in the cranial venous sinuses. (2) The paralysis tends to be more rapid and complete, and its distribution more accurately under con-

trol. The withdrawal of a considerable quantity of cerebrospinal fluid has no discernible constitutional effect on the patient.

The cannula, attached to the syringe, which is filled with the measured quantity of fluid to be injected, is gently inserted down the needle (care being taken not to alter the depth of the latter); the fluid is injected quickly and the instruments are as rapidly withdrawn. The cannula is long enough to project just beyond the point of the needle, so that, if the latter has only just penetrated the dura, the former will lie well within the theca; this explains the reason for withdrawing the needle to the greatest distance compatible with a free flow of cerebrospinal fluid.

The whole manœuvre occupies a very short time, and the child is immediately turned on his back, the head and shoulders well

FIG. 5.



Ready for operation after manœuvre shown in Fig. 4; X, lowest point of curve.

raised, and the back rounded by lifting up the legs (as shown in the accompanying illustrations, Figs. 1 to 7) for about fifteen to twenty seconds.

When the legs are brought down, the pelvis is raised on a folded towel to the extent which will regulate the height of paralysis required, and the immediate preparations for operation may be begun at once. The patient's face is screened from the sight of the operation by holding up the end of the towel, though, in many instances, this precaution is unnecessary.

Before dealing with the question of dosage, it may be well to refer to the unreality of a danger on which stress has, at times, been undeservedly laid—namely—*damage to the nerve roots* during the lumbar puncture. It has been stated that there is more risk of injury to the elements of the cauda equina with lateral than with

median puncture. I have elsewhere<sup>3</sup> shown how rare it is for one of the roots to be touched during lumbar puncture, for there is no mistaking the reality of such an accident, which almost induces a convulsion.

Again, an examination of the cauda equina shows how loosely the nerves float in the cerebrospinal fluid, so that even a deliberate attempt to wound them would, no doubt, often fail; for it is to be expected that they would slip to one side of the instrument, in a manner analogous to that by which distended intestines avoid injury.

*Dosage.*—It has been shown that absorption of drugs from the craniovertebral cavity is markedly delayed when these are kept in

FIG. 6.



Position for anaesthesia of legs and perineum.

the vicinity of the spinal theca; therefore, with a fluid which permits, to a great extent, such a control, the actual quantity of stovaine given is not, within reasonable limits, a factor of great importance as regards danger to life.

Under ordinary conditions, danger to life is to be feared from two sources: (*a*) Direct, from the spread of the paralysis to vital parts; (*b*) indirect, from absorption of stovaine into the circulation and the resulting poisonous effects.

The first method is controlled by *gravity*, the second by employing a solution of a very low diffusibility.

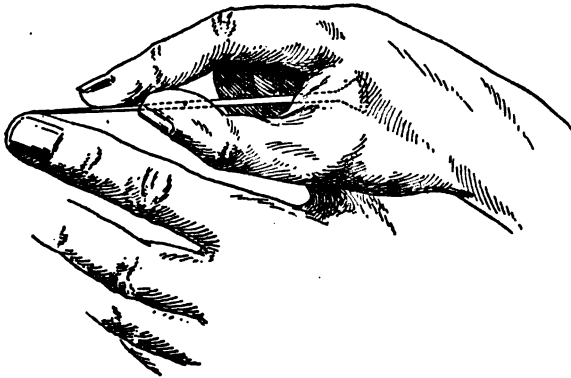
<sup>3</sup> *Lancet*, Sept. 25, 1909.

The fluid I recommend consists of dextrose 6 per cent., stovaine 3 per cent., suprarenin 0.005 per cent., in normal saline solution. With such a solution, a dose proportional to the required effect may be administered and repeated once, or even twice, should a prolongation of the anæsthesia be required. (I have even given 0.09 Gm. (1.4 grains) of stovaine to a child of about five years, in three successive injections for one long operation.)

The height of the paralysis is regulated by: (1) The *position* of the body (see illustrations); (2) *the bulk of the solution* employed, a larger bulk of fluid tending to produce a higher level of paralysis.

By repeated trials of different percentages of stovaine in dextrin,

FIG. 7.



Method of making the lumbar puncture.

the amount of the former has been fixed at the lowest figure compatible with satisfactory anæsthesia: dosage, therefore, is best considered in terms expressing the *bulk* of fluid rather than the actual quantity of stovaine to be administered.

Doses for children up to twelve years vary from 0.4 to 1.4 c.c. (6 to 23 minims); these quantities may be repeated during an operation should a prolongation of anæsthesia be required, and it is unnecessary, therefore, to give more than this in a single dose.

A smaller dose is required for a low than for a high paralysis; while a larger bulk of fluid will produce a longer duration of the anæsthesia. Thus if, in a child of about eight years, 0.8 c.c. (13 minims) dextrin-stovaine is administered for arthrectomy of the knee-joint, a satisfactory anæsthesia will result, but the effect of the

injection will not last so long as after the induction of a high paralysis with a larger bulk of fluid, *e.g.*, 1.2 c.c. (20 minims); for return of sensation progresses from above downwards, so that more time is required for it to reach the knee-joint with a high paralysis than with a low one, and a longer duration of anæsthesia results.

On a consideration of these laws we are in a position to make rules for the duration of anæsthesia. It is regulated: (1) By the *height* of the paralysis induced by means of gravity and the bulk of fluid; (2) to some extent by the *percentage of the stovaine*; and (3) by the *completeness* of the paralysis; for an incomplete anæsthesia (usually the result of faulty technic or faulty preparation of the stovaine) passes off more rapidly than a complete flaccid paralysis.

Again, two practical principles must be recognized to ensure success: (a) Not only must the paralysis include those segments which supply nerves to the skin area to be attacked by the operation, but it must involve the innervation of *all the tissues to be manipulated*. For example, it may be possible to perform the radical cure of an inguinal hernia with an anæsthesia extending to the eleventh dorsal segment, though it is likely that success will only be partial; for, when the sac is pulled upon, the traction will stretch the peritoneum above the paralyzed segment, and the resulting discomfort to the patient, though not amounting to actual pain, may make the child restless and interfere with the smooth course of the operation: under similar conditions manipulation of the great omentum will produce a like result. For the performance of orchidectomy, the anæsthesia should involve the abdomen at least to the umbilicus (tenth dorsal segment); for, otherwise, discomfort may result from traction on the testicle and the tissues of the spermatic cord. Other examples might be cited, but enough has been said to account in a measure for the partial successes reported from time to time; further, it seems not improbable that a lack of precaution in this respect may account for the persistence of views held, even at the present time, regarding the completeness of the anæsthesia: for some still maintain that analgesia only is to be obtained. The induction of analgesia only is explained on two lines: (1) No doubt the fault in technic just referred to is responsible to some extent for failure

to produce *complete anæsthesia*. (2) The highly diffusible solutions generally employed are the main cause of failure in this essential: reference will again be made to this subject. (b) In infants and very young children (especially in nervous subjects) it is wise to induce high anæsthesia, for thus the children are rendered more helpless, and, in the event of the onset of temporary disquietude or fear, management is simplified.

Now it has been shown that in the selection of the dose for administration we are to be guided by the following points: (1) The age of the patient; (2) the nature and situation of the operation; (3) the duration of the anæsthesia required; (4) the combination of the preceding points.

Though it is manifestly impossible to lay down strict injunctions for dosage, an average dose according to age may be useful. These doses, however, will have to be suited to particular cases, and modified to produce the desired effect.

AGE	DOSE
Under one month .....	0.4 to 0.5 c.c. ( 6 to 8 minims)
Under two years .....	0.6 to 0.8 c.c. (10 to 13 minims)
Under five years .....	0.6 to 1.0 c.c. (10 to 16 minims)
Under twelve years .....	1.0 to 1.4 c.c. (16 to 23 minims)

Before leaving the subject of technic, two further points require mention: (1) In cases of marked abdominal distention, great care must be exercised in regulating the height of the paralysis. For it must be remembered that in high anæsthesia, when a considerable degree of intercostal paralysis results, we have to trust to the diaphragm to carry out the greater part of the respiratory movements; the diaphragm is likely to be hampered by great abdominal distention and it is essential, therefore, to paralyze as few of the intercostal muscles as possible. In abdominal tumors encroaching on the thoracic viscera, some of the intercostal muscles must, of necessity, be paralyzed to ensure a satisfactory anæsthesia, but in such cases (when the patient is a young child) the risks of a carefully regulated spinal anæsthetic are considerably less than in inhalation anæsthesia, when the ultimate recovery of the patient (not only immediate recovery from operation) is hoped for. Again, in cases of great abdominal distention there is mechanical obstruction in flexing the spine to the full degree, which increases the difficulties of accu-

rate localization; it is wise, therefore, in such cases to aim at a slightly lower level of anæsthesia than that which is required, always remembering that the tendency is for a rather higher paralysis to ensue when the back cannot be well rounded. (2) The preliminary administration of bromides or morphine is not necessary as a rule. But, in older children, when a tedious operation is anticipated, the mental strain and associations of an operating theatre will be greatly minimized by such measures, while the combination of spinal anæsthesia with light inhalation anæsthesia (to the extent of unconsciousness only) is by no means to be deprecated in long operations involving a considerable degree of surgical shock.

**OBJECTS OF SPINAL ANÆSTHESIA.**—Briefly the objects of spinal anæsthesia are to substitute an efficient local anæsthesia, involving a large area of the body, for inhalation anæsthesia: (1) When the latter constitutes serious additional risks to, and therefore prejudices the success of, severe surgical operations; (2) in cases where inhalation anæsthesia is in itself a serious danger, owing to the precarious condition of the patient; (3) when inhalation anæsthesia is definitely contraindicated; (4) in some cases where the increased facilities in operative manipulations afforded by spinal anæsthesia are of sufficient importance to justify its substitution for the older method.

To amplify these four points:

1. Whatever views we hold as to the nature and origin of surgical shock, it must be admitted that its onset is determined either directly or indirectly, by the manipulations of the operation. True it is that the phenomena of shock are no doubt precipitated and accentuated by other factors, such as collapse from hemorrhage, toxæmia or the effect of inhalation anæsthesia, exhaustion due to loss of heat from the necessary exposure, and prolonged mental strain or excitement; these, however, are factors which, though important in themselves as influencing the degree of collapse in a given case, demand an independent line of preventive treatment, and are to be considered entirely apart from true surgical shock induced by the steps of the operation.

If now it be possible to cut off completely the part of the body to be operated upon from every nervous connection above this re-

gion, we have a right to expect a considerable modification of the immediate constitutional effects produced by a severe operation. It has elsewhere been shown how I have endeavored to produce this effect by concentrating the action of the stovaine (by means of eliminating, as far as possible, the diffusion, and consequently the dilution of the drug) on a small area of the nerve roots at the desired level, in order to create a complete "nervous block" to centripetal impulses, akin to a transverse lesion of the spinal cord. Evidence, both clinical and experimental is forthcoming to show that, in all probability, this condition of affairs actually does exist; if this is so, we have a very powerful preventive against true surgical shock in operations below the thorax. Experimental evidence I am not at liberty to submit at present, as the researches of Dr. L. G. Parsons and the writer on this subject are, as yet, incomplete; clinical evidence has been brought before the profession on several occasions to show that small children may be submitted to severe operations without evincing any evidence of collapse, which is such a dreaded symptom of profound surgical shock. The results of severe and emergency operations (particularly within the abdomen) have so greatly improved at Great Ormond Street, since the adoption of spinal anæsthesia in these cases, that the influence of this method cannot be doubted. All the evidence so far accumulated by Dr. Parsons and the writer supports the view that, so long as the anæsthesia is *complete*, surgical shock is practically eliminated; but that symptoms will commence to manifest themselves as the effect of the drug wears off, and anæsthesia passes into analgesia.

I would submit that when, in due course, definite proof can be adduced in support of these views, the *strongest argument possible* will have been presented in favor of the adoption of spinal anæsthesia in this class of cases, particularly at the extremes of life.

2. The second class of cases embraces patients exhausted by illness or poisoned by toxins; the vital centres of such subjects, already depressed to a dangerous extent, can ill submit to the additional "toxin" of inhalation anæsthesia for an operation of necessity, be it trivial or severe; in the latter case the shock of the operation is superadded. To this group belong acute infections of the peritoneal cavity, the various forms of intestinal obstruction, some cases of abdominal tuberculosis, amputations, excisions of



joints and extensive operations on bones in children who are exhausted by long illness and hectic fever, etc. Space forbids a more minute account of these cases, but, with these few principles as a basis, further details may be left to the experience of the surgeon.

3. It may be assumed that inhalation anæsthesia is definitely contraindicated in subjects of phthisis, renal insufficiency in all its forms, and uncompensated heart disease (especially when accompanied by bronchitis or renal congestion). I have not employed spinal anæsthesia in uncompensated heart disease, but should not hesitate to do so in operations of necessity. Whether this method is a satisfactory alternative in renal insufficiency is at present open to question, though it is certainly deserving of a trial.

Subjects of "cyclic vomiting" should not be submitted to a general anæsthetic, but spinal anæsthesia should be employed whenever it is applicable. In the present state of knowledge the so-called status lymphaticus can hardly be brought into the discussion.

4. The increased facilities afforded to the surgeon by spinal anæsthesia are diminished hemorrhage and perfect muscular flaccidity, both of which (especially the latter) add very greatly to the rapidity of an operation, a consideration of some moment when speed is essential to the patient's welfare. Thus a small incision permits of easy access to parts by wide retraction of the wound, for which a far larger incision would be necessary under general anæsthesia, a feature of great value in abdominal exploration. The anæmia and flaccidity of the bowel wall facilitate manipulations of the intestines; in intussusceptions, particularly, reduction is proportionately simplified, an advantage admitted by many surgeons. Finally mention may be made of the advantage of this muscular flaccidity in the apposition and fixation of the fractured ends of long bones by operation, spinal anæsthesia proving itself, at times, a distinct aid to a difficult operation. This aspect of the subject has been fully dealt with in previous papers.

**LIMITATIONS OF SPINAL ANÆSTHESIA.**—Since, by this method, a complete flaccid paralysis of the parts involved is the essential feature, it follows that anæsthesia above the sixth cervical segment is incompatible with life. Paralysis of the intercostal muscles is feasible provided the diaphragm is intact, and, indeed, the involvement of the lower intercostals is necessary for efficient anæsthesia

in operations in the upper part of the abdomen and under the diaphragm. Operations within the thorax should be performed under general anæsthesia; spinal anæsthesia is inadmissible here, for the embarrassment of respiration consequent on opening the pleural cavity would only be enhanced by the necessary intercostal paralysis. It follows, therefore, that this method is applicable only to surgical procedures below the diaphragm, and it may be employed for any operation below this level in suitable cases when there is no contraindication.

It is, of course, possible to perform operations within the thorax, and on the head and neck, by means of highly diffusible fluids (with a careful regulation of the dose) administered to produce analgesia only. It seems that such methods have very little advantage, if any, over general anæsthesia in the control of surgical shock, while they are open to the objection that drugs, so introduced, are rapidly absorbed into the blood stream. Again, it is quite a question how far such analgesia is due to a direct local effect in the theca, and how far it results from the circulation of the toxic agent in the blood-stream after rapid absorption, which we know takes place under these conditions. Though I can at present offer no definite proof of these views, if the latter method plays any part in the production of analgesia, the same conditions prevail as in inhalation anæsthesia with this important exception: *there is no control over the effects of the anæsthetic*. In this case the method cannot be held either safe or advantageous. I am considering, therefore, only methods aiming at the production of a complete flaccid paralysis, and these should not be employed in operations above the diaphragm. It has been stated that the greatest care should be exercised when abdominal distention is marked, and, in malignant tumors of the kidney, or in this vicinity (especially if encroaching on the thoracic viscera), abdominal distention is a definite contraindication to spinal anæsthesia, particularly when free fluid is present in the peritoneum.

Spinal caries, adhesions within the spinal theca such as sometimes occur after recovery from meningococcal meningitis, hydrocephalus, and other conditions associated with an increase of intracranial pressure, are all unsuitable and may prove fatal. Finally, in operations on tendons for the remedy of spastic contractions,

spinal anæsthesia is not suitable; for the resulting flaccid paralysis renders it difficult to judge the requisite amount of lengthening or shortening.

I have endeavored, in these few pages, to present some points which may be of assistance in the judicious selection of cases. Failure in this respect has, no doubt, been responsible to a certain extent for a death rate, which, however, has decreased enormously as a knowledge of the subject has advanced. Faulty technic, also, has led to failures which will similarly decrease in number with the increase of proficiency.

Spinal anæsthesia will always be of value in those rare emergencies when no anæsthetist is obtainable; but its main feature is undoubtedly the marked influence it seems to exert in the prevention of surgical shock, and I believe that it will stand or fall as a recognized procedure in the future, according as it fulfils or falls short of this important requirement.

# **SURGICAL TUBERCULOSIS OF JOINTS AND THE EFFECTS OF SURGICAL TREATMENT \***

**BY JAMES K. YOUNG, M.D.**

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## **TRACTION AND FIXATION**

For many years there has been a discussion in regard to the relative merits of fixation and traction in regard to treatment of tuberculosis of the joints. The effect of traction upon a tuberculous joint is to quiet the muscles, relieve the spasm, and diminish the amount of friction and irritation between the joint extremities. Its effects have been demonstrated by the comparison of a series of cases treated in different institutions in which traction was used and in which traction was omitted and the evidence is overwhelmingly in favor of the use of traction in the treatment of tuberculous lesions of the joints. The advocates of fixation also emphasize the rest of the part, the relief from friction, and the recovery of the inflamed joints by the use of this method of treatment. The X-ray examination of the patients treated without traction and with fixation demonstrated the fact that there is more destruction in joints from the use of fixation alone than where traction is given. The most satisfactory results in my hands have been obtained from the combination of these two methods of fixation and traction. This is well demonstrated in the hip, where by the complete fixation of the hip-joint the body and the entire lower extremity, including the foot, combined with the use of traction, furnish an ideal mode of treatment.

## **TREATMENT OF ABSCESSSES**

The surgical treatment of abscesses becomes much more exact and satisfactory if we know the character of the infection. For several years past I have been in the habit of making diagnostic punctures of all collections of pus which have come under my observation. This can be readily accomplished by means of an ordinary hypodermic syringe; a small quantity of the pus only is needed for bacteriological examination and culture. A portion of this fluid can be stained at once and examined by the microscope, and the other portion can be cultured or if necessary inoculation of guinea-

pigs can be made. By this method we are able to determine whether the collection of pus in a case of tuberculosis of bones and joints is sterile in character, whether it is tuberculous, or whether it is due to a mixed infection with some one of the diplococcic group of bacteria. The variety of germs found in these collections is astonishing, and furnishes a key to the different methods of surgical treatment. If the collection of pus is sterile (Fig. 1) the abscesses can be freely incised and curetted and then closed without drainage; if it contains tubercle bacilli the abscesses can be incised and thoroughly curetted and closed without drainage by a double row of sutures, a deep and superficial layer; if the collection has been infected with the pyogenic organisms it should be drained for a considerable period and at the time of the operation it can be treated with antiseptic applications. If the germ found be a rare one, as *Actinomyces*, or, as in one rare instance, the bacillus of hog cholera which was found associated with the staphylococcus, the exact character of the disease would be recognized.

#### SEROTHERAPY

When the abscesses have become infected with pyogenic organisms and this has produced a constitutional effect, it will be necessary to treat the patient by the use of autogenous vaccines; the pus from abscesses or sinuses is cultured and a vaccine prepared from this and injected beneath the skin. The effect of these injections upon a patient infected from an abscess is marked and characteristic, the clinical symptoms subside and the patient is again restored to normal condition. In all instances where it is practicable or possible the opsonic index should be used in connection with the serotherapy. There is a tendency at the present time to depend upon the clinical symptoms and to omit the use of the opsonic index introduced by Wright, but I am convinced that notwithstanding the time and expense involved in this method it should always be employed.

#### ERASIONS AND EXCISIONS

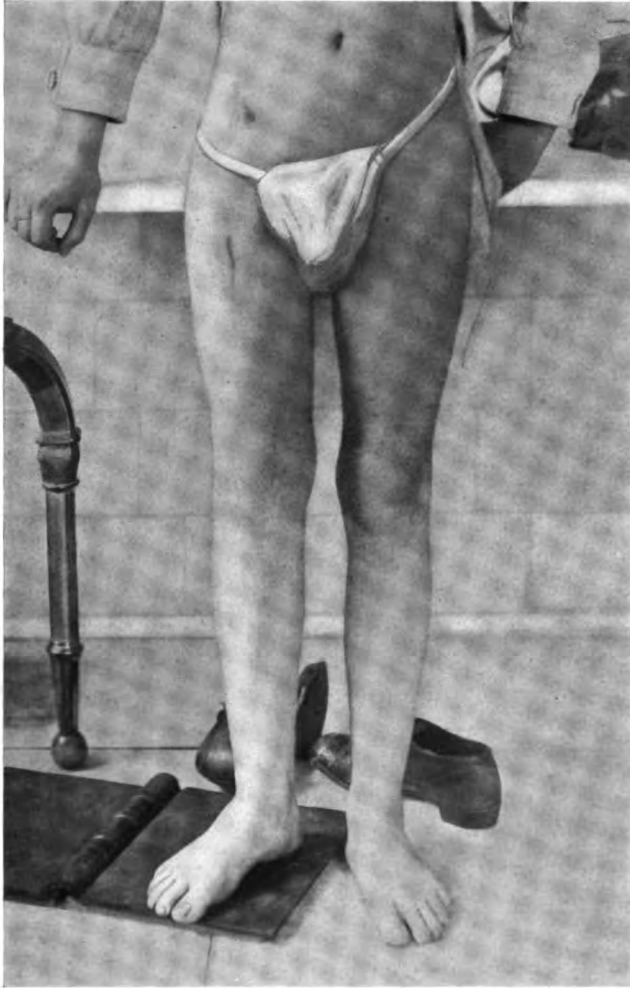
The use of formal excisions in the treatment of tuberculosis of the joints is not so frequent to-day as formerly, because cases are treated earlier and, as many of them occur in children, formal excisions are avoided wherever possible. In adults it is necessary to perform this excision of the articulation because of the involvement

**FIG. 1.**



**Lumbar abscess the culture of which was sterile.**

**FIG. 2.**



**Hip disease showing sinuses infected with staphylococcus.**

of extensive portions of bone, and growth of the part will not be so greatly interfered with. It is also necessary in adults to perform these operations because very often it is a choice between an excision and an amputation of the part. By erosion of the joints, as introduced by Professor Willard, we mean the removal of those portions of the joints which are diseased, leaving the healthy tissues intact. This is of the greatest service in children where the removal of the diseased portion leaves only the epiphyseal line intact and does not interfere with the growth of the length of the bone. At the time of the erosion of the part the member may be improved in appearance, the joints may be straightened; these erosions may be repeated from time to time as they become necessary and other portions of the joints are involved, by this method by the use of erosion in children many useful limbs are saved which otherwise might be sacrificed. The improvement of the patient's clinical symptoms by erosion is often very marked, the temperature improved, the leucocyte count diminished, the hæmoglobin increased in percentage, and the general appearance of the patient greatly improved. After erosion of the joints free drainage can be established and the cavities can be treated from time to time.

#### THE TREATMENT OF SINUSES

Before beginning the treatment of sinuses connected with tuberculous bone and joint disease, a bacteriological examination should always be made, as in this way only can the character of the discharge be determined. If the sinus is infected, as it is in a large number of cases (Fig. 2), it should be thoroughly curetted and should then be irrigated with tincture of iodine or iodoform, or carbolic acid and alcohol, or some similar method. If the sinus is found to be sterile injections of bismuth paste will be found of great value. The bismuth paste appears to act in these sinuses by its mechanical pressure as well as by its chemical action, the pressure of the bismuth increases the cell formation and the action of the nitrites is beneficial upon the wall of the sinus. It is a serious mistake to inject bismuth paste into a long sinus which contains pyogenic organisms of different characters. The result of such injection is to block the opening and cause the absorption of the pus, and the result is a sapræmia which at times is quite marked and occasionally is fatal.



## A CLINICAL LECTURE ON THE RETROCÆCAL APPENDIX

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AN appendix lying behind the cæcum must receive at the hands of the surgeon special consideration, both in regard to the manner of removing it, and in regard to the pathological changes which may follow such a position. In the early days of the development of the subject of appendicitis it was thought that the most frequent position in which the appendix was to be found in the abdomen was pointing inwards, or downwards and inwards, from the end of the cæcum. Further investigations during operation and upon cadavers showed that this downward and inward position of the appendix was found only in about sixty per cent. of the cases. Of the remaining forty per cent., over thirty are represented by an appendix located behind the cæcum, usually pointing directly upwards toward the liver, less frequently upward and inward.

In the case in which I have just operated this retrocæcal position of the appendix could be readily recognized upon opening the abdomen by the fact that the ordinary rule, which is followed by the majority of surgeons in finding the appendix, namely, to follow the anterior longitudinal band of the cæcum downward, failed at first to find the appendix. This rule of following the anterior longitudinal band of the cæcum to find the appendix cannot be too strongly emphasized. I have frequently witnessed operations in which search was made for a long time, in one case for over half an hour, without finding the appendix. Such a fruitless search could not occur if the operator would follow this band, as it invariably leads to the appendix, even though the latter be buried in adhesions. The retrocæcal appendix can be best found by the following rule: When the abdomen is opened by the McBurney, or right rectus incision, according to the preference of the individual operator, the

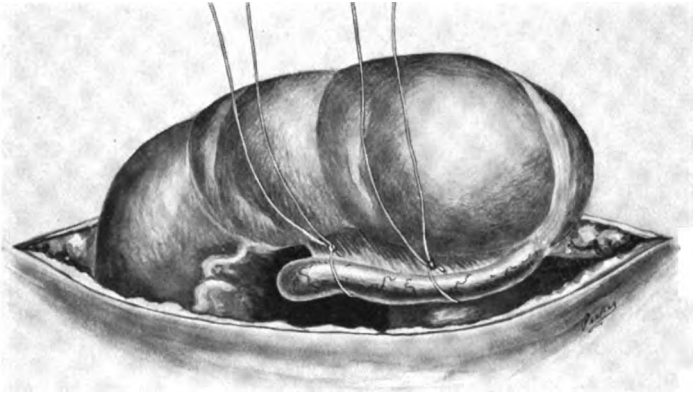
finger is swept around the end of the cæcum in order to find the appendix. Should none be found, it is advisable to lift the cæcum through the wound and follow this anterior longitudinal band to its end. At times, the first or proximal portion of the retrocæcal appendix can be readily distinguished at the end of the band, because it has in some cases a relatively long mesenteriolum which permits it to be lifted away from the cæcum. In other cases the mesenteriolum is so short that the appendix almost seems to be a portion of the posterior wall of the cæcum. In such cases I have found it very useful (and, in fact, in almost every variety of retrocæcal appendix) to utilize the method shown in the accompanying illustration (Fig. 1). When the cæcum is brought into the wound, a fairly strong catgut suture is inserted through the mesenteriolum and tied close to the appendix, but not so tightly as to constrict the latter. This "guy-rope" permits the cæcum to be lifted up into the wound in such a position that the retrocæcal appendix can be gradually delivered with the aid of one to three of these sutures. I have found this method invaluable in a large number of cases of such retrocæcal appendices, especially when the appendix could scarcely be recognized as such, being covered by plastic exudate or older fibrous adhesions in such a manner that one could only recognize it as an appendix by palpating a firm cord-like structure which began at the end of the anterior longitudinal band, and then could be gradually stripped from its bed by the use of a Kocher dissector. After the retrocæcal appendix has been delivered into the wound, if the mesenteriolum is very short, and, as is often the case, extremely vascular, a very great saving in time and annoyance consists in ligating the appendix at its base and then proceeding to separate it from the posterior wall of the cæcum by working from its base towards the tip, instead of the reverse, that is, the tip towards the base, as is done in the majority of appendectomies. This method of ligating the base close to the cæcum will often save fifteen to twenty minutes time and much bleeding from the adhesions and vessels of the mesenteriolum. I have used this reversed method of removing the appendix a number of times in other than retrocæcal appendices and find it an extremely valuable aid.

When abscess formation has occurred around the retrocæcal appendix it can cause (as in Fig. 2) such an amount of contraction

of the psoas muscle as to simulate to perfection a perinephritic suppuration, or even hip-joint disease.

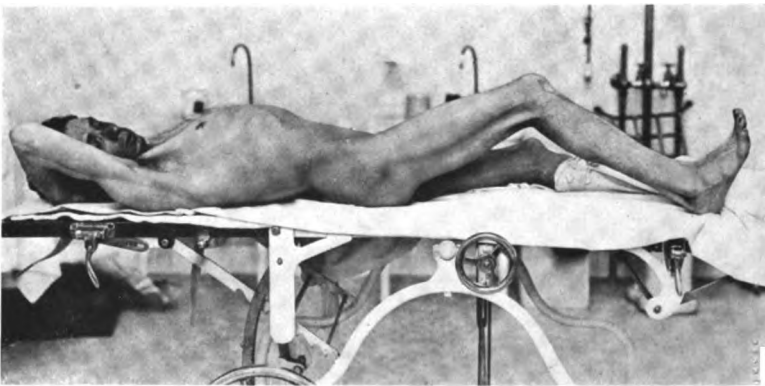
The second case which I show you (Fig. 2) is that of a young man, who entered the hospital with his thigh flexed upon the abdomen, marked rigidity in the region of the hip-joint, great pain on effects at extending the limb, a temperature of 39° C. (102° F.) and a history of sudden onset of pain just above the crest of the ilium, that is, in the right iliocostal space. Examination showed that movements of the hip-joint itself were not restricted, with the exception of flexion, so that coxitis could be excluded. The urine was negative. There was no history which pointed to any renal infection or calculi as the cause of the perinephritis. The spastic flexed position of the thigh pointed to some suppuration in the vicinity of the psoas muscle. From the absence of tenderness over the region of the kidney proper, and the presence of rigidity over the right half of the abdomen, a diagnosis of suppuration of a retro-cæcal appendix with irritation of the psoas muscle was made. The abdomen was opened through a right rectus incision. As is so frequently the case in abscess formation around the retrocæcal appendix, the cæcum presented into the anterior abdominal wound almost immediately upon opening the peritoneal cavity. This forward displacement of the cæcum was at once seen to be due to a mass lying behind it, extending from the iliac fossa to about the middle of the ascending colon. I call attention to this special mode of abscess formation in retrocæcal appendicitis, for the reason that it is not as frequently described as it deserves to be. Such an abscess requires special treatment. In order to evacuate the pus lying in such a retrocæcal abscess, it has always been my custom to displace the cæcum gently inwards, wall off, as far as possible, the general peritoneal cavity by packing in strips of gauze lightly, and then proceed to open the abscess by the use of some blunt instrument, like a blunt-pointed curved scissors, along the outer side of the cæcum just where the peritoneum is reflected upon the posterior surface of the cæcum. After evacuating the pus from the abscess cavity, it has been my routine practice in these cases of retrocæcal appendiceal abscess, to make a diligent search for the appendix and remove it. I have recently performed twelve secondary appendectomies for retrocæcal appendices. I call these secondary appendec-

FIG. 1.



"Guy-rope" method of delivering a retrocaecal appendix.

FIG. 2.



Spastic rigidity of the psoas muscle from appendicitis.

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tomies where such a retrocæcal abscess was opened without the removal of the appendix at the primary operation, and the symptoms either recurred, or a fistula persisted which required secondary removal of the appendix. It is not advisable, however, to risk breaking down adhesions in order to attempt to remove such an appendix if the cæcum is situated at a level higher than normal, so that the appendix lies just in front of the kidney. These retrocæcal appendiceal abscesses also require special consideration in regard to drainage. They are best drained by making a counter-opening just above the crest of the ilium and inserting a drainage tube, well protected by several layers of gauze and gutta percha tissue from exerting too great a pressure on the posterior aspect of the cæcum and the stump of the appendix. I have frequently seen fecal fistula and ventral hernia following attempts to drain such retrocæcal abscesses entirely through the original abdominal incision. I have also made it a rule to place patients, who have had retrocæcal appendiceal abscesses, in the Fowler position after operation, owing to the close proximity of the abscess cavity to the subphrenic space. In an article upon subphrenic abscess following appendicitis, I called attention to the frequency with which retrocæcal appendiceal abscesses are followed by suppuration in the subphrenic space. This is due to the fact that the pus organisms can so readily migrate along the outer side of the cæcum and ascending colon into the subphrenic space. Whenever a patient who has had a retrocæcal appendiceal abscess begins to have persistent and high temperature following the draining of such an abscess, one should first look for evidences of retention in the original abscess cavity, and if retention is not found, it is best to consider exploratory puncture of the subphrenic space, or look for evidences, such as a convex area of dulness above the ordinary liver dulness, which is often present in a subphrenic abscess.

## THE MANAGEMENT OF FOUR KINDS OF APPENDICITIS

BY ROBERT T. MORRIS, M.D.

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THE commonest kind of appendicitis we will consider first. This is the form associated with normal involution of the appendix. It is an irritative lesion and not an infective lesion.

Normal involution of the appendix may begin at any time in the life of the individual; but we seldom find demonstration of symptoms before the patient is twenty-five years of age, and most of the patients will be older than that. The appendix undergoing normal involution becomes smaller and harder, as its various structures are gradually replaced by connective tissue, and the lumen disappears little by little.

Appendices presenting these changes of fibroid degeneration are the ones commonly included in the statistics of observers who try to prove from post-mortem findings that more than half of the people suffer from infective appendicitis at some time in their lives. Writers not recognizing fibroid degeneration as it belongs to normal involution have given misleading impressions. An entirely different form of fibroid structure is found in appendices which have been the seat of infective appendicitis. The appendices undergoing normal involution, retain nerve filaments for a long time. The mesappendix becomes shorter and shorter and may disappear entirely, finally leaving the remains of the appendix under a single flat layer of peritoneum. The fibrous appendix representing the scar following infective appendicitis is quite different in character. It has usually lost the nerve filaments which persist in the normal involution appendix. It may or may not retain a mesappendix, and it is often surrounded by old adhesions beside containing one or more mucous inclusions.

The symptoms belonging to the scar appendix following infective appendicitis are seldom more than ordinary adhesion symptoms,

but the symptoms belonging to normal involution of the appendix are much more important. They depend upon the irritation of nerve filaments entrapped in contracting connective tissue, and they are of two chief sorts. The first set of symptoms belongs to the sensory nerve filaments. Such filaments entrapped in the contracting connective tissue of the appendix undergoing normal involution, give rise to sensations of discomfort in the appendix region. Sometimes there is actual pain, and tenderness on pressure in the appendix region. More often the sensation is that of a burning or a dull ache, coming on from time to time, sometimes absent for days, and then causing discomfort for several days in succession. Sometimes there is only a fleeting "neuralgic twinge" lasting for a moment and then forgotten by the patient.

The second set of symptoms belonging to the fibroid change of normal involution of the appendix is usually more important. Irritation of the sympathetic nerves leads to reflex irritation of the intimate ganglia of the bowel wall, and we have in consequence derangement of bowel function. The patients come to the doctor for treatment for "chronic intestinal dyspepsia" and that condition is often enough treated as a diagnostic entity, without having the rôle of the appendix noticed at all. In cases in which fibroid degeneration of the appendix is suspected to be the cause for "chronic intestinal dyspepsia" we make the diagnosis as a rule upon three lines of evidence. The first point in evidence consists in finding the right group of lumbar ganglia hypersensitive on deep pressure with the finger, at a place about an inch and a half to the right of the navel, close to the spinal column. The next point consists in finding the cæcum and ascending colon distended with gas. The third point is discovered on finding the appendix harder than normal to the touch on palpation.

How are we to manage cases of fibroid degeneration of the appendix? The symptoms and various disturbances last over a term of years, and management is to depend upon the severity of the symptoms. If the chronic intestinal dyspepsia is amenable to ordinary hygienic and dietetic treatment, with harmless use of drugs, and if the patient does not need to make that treatment his chief object in life, we may advise against any surgical procedure. When medical treatment does not suffice, it is a comparatively easy



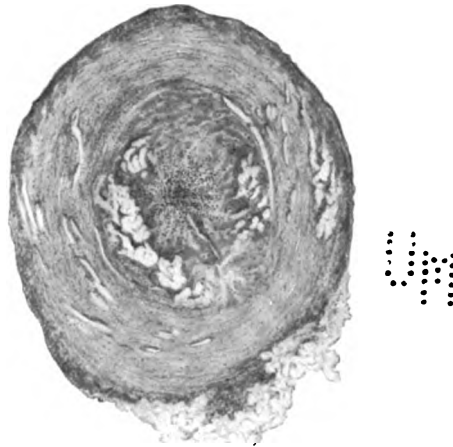
matter as a rule to remove the appendix; for adhesions are not present to complicate the work, and we apparently have an established local hyperleucocytosis due to the chronic local irritation which is protective against infection at the time of operation. This leucocyte protection apparently guards against the development of ordinary infective appendicitis as well, and added to disappearance of the structures susceptible of infection, has led me to name this commonest kind of appendicitis "protective appendicitis." (Fig. 1.)

The next form of appendicitis in order of frequency of occurrence is the one which is usually carried in mind when the word "appendicitis" unqualified, is employed. This is appendicitis with intrinsic infection, and the reason why a form occurring less frequently should be spoken of generically to cover the whole subject is because the symptoms are so much more violent in character and the disease so much more destructive when it does occur. Appendicitis by intrinsic infection appears to depend upon any cause for rapid swelling of the soft inner coats of the appendix to a point of compression anæmia, within the narrow, tight external sheath of peritoneum and muscularis. The tissues which are anæmic by compression are then readily attacked by bowel bacteria, having temporarily lost their power of calling out local protectives through the blood and lymph circulatory systems.

When a patient has protective appendicitis—the commonest form—we may deliberately leave the question of operation to his wishes. When the patient has intrinsic infective appendicitis progressing, the question is not one that can be left to the wishes of the patient, or even to the judgment of consultants. We must operate just as soon as the diagnosis is made. The reason why the question cannot be left to the wishes of the patient or to the judgment of consultants is because the course of intrinsic infective appendicitis, at all times, lies outside of the limitations of judgment. There never will come a time when experience or knowledge on the part of consultants will allow of anything more than a guess as to the outcome, and the patient has to stand the result of such guessing. Our operative death rate is now so small, at the hands of many surgeons, that the management of intrinsic infective appendicitis is not one allowing of much discussion.

The third form of appendicitis in frequency of occurrence is

FIG. 1.



Microscopical appearance of fibroid degeneration of the vermiform appendix: transverse section, showing replacement of the inner coats by connective tissue.



like protective appendicitis, an irritative lesion, and not an infection. This is syncongestive appendicitis. When there is swelling of the appendix along with swelling of neighboring bowel, the process usually occurs so gradually that all structures of the appendix become distended evenly with serous interstitial infiltrates, and the patient avoids the disaster of rapid swelling of the inner coats alone. Syncongestive appendicitis is often found with so-called "lithæmic" swelling of the bowel wall, and occurs in rheumatic or gouty patients. It is in evidence with cases of loose right kidney, depending according to Edebohls upon pressure of the kidney on the superior mesenteric vein with congestion of the cæcum and appendix in consequence. Syncongestive appendicitis is also a secondary feature of lymph obstructions which end in ascites.

With this type of appendicitis we have more or less pain and tenderness referable to the appendix region, and there is hypersensitiveness of the right group of lumbar ganglia. Management depends upon the cause. In "lithæmic" cases the use of salicylates and other ordinary resources will result in diminution of the appendix symptoms. Medical treatment and not surgical is called for in the syncongestive group of cases.

The fourth kind of appendicitis is another infective lesion. Appendicitis by extrinsic infection depends upon extension of infection from some neighboring structure, as from the oviduct, or from a tuberculous peritoneum. Swelling ordinarily progresses so slowly, with the external coats becoming swollen first, that the patient may avoid the consequences of rapid swelling of the inner coats primarily. There are cases of extrinsic infection in which the appendix feature becomes dominant, however, with symptoms similar to those of intrinsic infective appendicitis. In many of these cases we are to operate as soon as the appendix feature is distinctly the one calling for attention.

## THE "ACUTE ABDOMEN" IN CHILDREN, WITH SPECIAL REFERENCE TO ACUTE INTUSSUS- CEPTION AND ACUTE APPENDICITIS

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pital for Women, Edinburgh

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IT is a fact which has frequently been stated that the surgery of the abdomen in children is limited in its field. While this may be true as far as the more chronic conditions are concerned, as compared with the adult, the acute conditions requiring immediate operation are in all probability as frequent as in the adult. During the last five years almost 200 emergency operations have fallen to my lot as an Assistant Surgeon to the Royal Hospital for Sick Children, and as these represent about one-third of the total number of cases of "acute abdomen" operated upon and passing through a Surgical Department of 40 beds, an average of 10 per cent. does not overestimate the frequency of this class of cases in the wards of a children's hospital.

From a record of these cases, therefore, it is interesting to analyze them from the point of view of the causation of the acute abdomen in children, and the age incidence of the various conditions.

For convenience of reference, I might state that the cases incorporated include the following:

1. Acute intussusception (30 cases).
2. Acute appendicitis (102 cases).
3. General peritonitis (streptococcal, pneumococcal, etc.).<sup>1</sup>
4. Acute intestinal obstruction.
5. Strangulated hernia.
6. Congenital malformations.
7. Various twisted ovarian tumors; Meckel's diverticulum.

*Acute intussusception* was responsible for one-sixth of the cases herein recorded. This condition is the cause of acute abdominal

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<sup>1</sup> The remaining conditions will form the subjects of a later contribution.

symptoms necessitating operation in 90 per cent. of cases under one year of age. It occurs in healthy, well nourished children, whether breast-fed or bottle-fed, with a slight preponderance in favor of the breast-fed. Males seem to be slightly more liable to the condition than females, in the proportion of 17 to 14 in the present series of cases, but records of other observers indicate a still greater prevalence in the male sex. The youngest child submitted to operation was 10 weeks old, and the oldest a boy of 5 years. Most cases occurred between the fifth and tenth month of life, during early dentition, and the seventh month especially would seem to be the period of greatest prevalence.

The symptomatology in these cases is practically constant, the severity of each symptom depending first on the duration of the condition, and secondly on the extent of the invagination. The cardinal symptoms in these cases were the sudden onset of colicky pain, as shown by the pained expression of the face, drawing up of the legs, and the sharp cry on wakening out of sleep characteristic of these cases. These symptoms taken along with the mucohemorrhagic discharge from the rectum and the presence of a tumor associated with vomiting lead one to an almost certain diagnosis of the condition when they are present in an otherwise healthy child. All these signs may however be present along with a tumor and yet no intussusception is present. They may all be absent and yet an invagination is present.

In my experience, there is one sign which never fails to indicate the presence of an intussusception, and that is the presence of blood and mucus in the rectum. In an infant under one year, it is rare to have this sign in ileocolitis, a condition in older children giving rise to a similar discharge from the bowel, but of a more watery nature. If on passing one's finger into the rectum such a discharge is present, this alone, in the absence of all other signs including tumor, is in my opinion an indication for surgical interference.

In four of these cases, no tumor could be palpated, but it was revealed at operation. This generally occurs when the intussusception has not passed beyond the hepatic flexure or transverse colon and becomes hidden under the liver.

The tumor may be felt anywhere. Where the intussusception is small, it is generally felt under the upper part of the right rectus,

or according to its extent, into the left hypochondriac or iliac regions, or in the rectum itself.

It may be of interest to give the position of the apex of the intussusception in the present series of cases.

It had reached the hepatic flexure in 5 cases, transverse colon in 3 cases, splenic flexure in 5 cases, pelvic colon in 15 cases, and rectum in 3 cases. Although the extent to which the intussusception passes round the colon depends in a certain degree on the duration of the invagination, the relation between the two is not constant.

Thus in one case, that of a female child of 7 months, in whom symptoms had been present 3 days, the intussusception was a small one, two inches in length, extending to the hepatic flexure of the colon, whereas in another case of 12 hours' duration the intussusception had extended well into the pelvic colon.

It is interesting to note, however, that in the former there was a tight impaction causing considerable difficulty in reduction, whereas in the latter the reduction was easily accomplished; so that the size of an intussusception is no indication of the length of time of the invagination, or the ease with which it may be reduced. The extent of the invagination also does not seem appreciably to affect the mortality, as this is influenced chiefly by the duration of the condition, and the ease or difficulty of reduction. The condition of the abdomen is of interest in these cases. In the majority of cases it is tense and palpation is resisted by tightening of the abdominal muscles, the respiratory movements however being free. The tumor is felt, but generally through a tense abdominal wall and is indefinite in its outline.

It occasionally happens that the infant's abdomen is lax and flabby. The tumor can almost be grasped in the palm of the hand, and the anterior abdominal wall can be pressed against the spinal column with ease. This occurs in two classes of cases. It is seen where the intussusceptum slips into and out of the intussusciens cavity, where there is no congestion or œdema of the bowel or its mesentery. It is seen therefore in early cases, and especially in those in which the condition is recurrent and reduction may occur spontaneously. This rare condition of the abdomen is seen, however, in advanced cases where the child suffers from severe shock, and the most marked degree of laxity of the abdomen I have seen

was in such a case where reduction was difficult and the child died subsequently.

It is a striking proof of the improved diagnostic powers of the general practitioner in recognizing this condition and of his promptitude in recommending operative treatment, that such a large proportion of cases are admitted to hospital within 24 hours of the onset of symptoms. The operation was performed within 24 hours in 14 cases and 48 hours in 7 cases, the remaining cases being admitted on the third, fourth, and fifth days of the disease. Of those operated upon within 24 hours 13 out of the 14 recovered—mortality 7 per cent.; within 48 hours, 6 out of the 7 recovered—mortality 14 per cent.; and of the remaining 10 cases 5 died—mortality 50 per cent.

I have no hesitation in saying that early diagnosis is of the utmost importance, and the only treatment offering uniform success is that by operation.

To anyone who has operated upon a series of these cases, and, I believe, to any practitioner witnessing these operations, it seems incredible that such methods as fluid or gaseous distention of the colon should be recommended. One must admit that such means as these may and probably do partially reduce the invaginated bowel, but the difficulty which is experienced in so many cases in manually reducing the last inch or two of the intussusceptum is in itself a proof of the fruitlessness of any gaseous or fluid pressure being sufficient to bring about reduction without rupture of the bowel. It is sincerely to be hoped that colonic distention as a method of treatment will soon be looked upon as a relic of the past except under circumstances making operation impossible.

The type of invagination in these cases was ileocæcal, except in two cases, in which the intussusceptum was double, the cæcum remaining uninvaginated. In one of these the invagination began in a Meckel's diverticulum. In one instance an ileocæcal intussusception recurred after one month, the type on the second being ileocolic. Immediate operation was performed in all these cases, and reduction by manipulation succeeded in all but two cases. In one case the intussusciens was incised longitudinally, the head of the intussusceptum delivered and resected and the bowel closed by Maunsell's method. In the other the intussusception was left outside the



abdomen, and a Paul's tube inserted into the small intestine. Secondary resection was performed on the 17th day. Both these cases died within 24 hours of resection.

It is interesting to note that during reduction in the successful cases, the cæcum and vermiform appendix with its mesentery were the last to appear, but in 10 cases there remained after their reduction a marked cæcal dimple generally large enough to admit the point of the finger. This was situated at the apex of the cæcum in 8 cases, and in the remaining two on the lateral wall opposite the mesenteric attachment.

It is not the purpose of this paper to discuss the etiology or mechanism of this condition, as this has been thoroughly investigated by Dunbar, Fitzwilliams, d'Arcy Power, Clubbe and others. Death resulted in 7 cases. Of these 3 died within 24 hours of shock. Two cases died 10 days and 19 days afterwards of slow progressive general peritonitis due to *Bacillus coli* infection. Both were severe cases and the bowel was much congested and cyanosed. Another died of postoperative ileus, due to paresis of the bowel. One death was due to delayed chloroform poisoning, in which the child received pure chloroform three times within 24 hours; once by the family attendant, once by the resident surgeon on admission, and lastly at operation.

This is a matter of grave consideration, the repeated administration of chloroform to young infants within a short space of time, nor do I believe it justifiable in order to demonstrate the presence of a tumor unless operation is to follow immediately.

The rectal examination showing the presence of blood and mucus in the lower bowel is in my opinion sufficient for the diagnosis of the condition, without of necessity demonstrating the presence of a tumor.

*Acute Appendicitis.*—This as in the adult is the commonest cause of acute abdominal symptoms. Boys suffer much more frequently than girls in the proportion of 65 to 40, and the age of greatest frequency appears to be in both sexes between 6 and 8 years. This may be in part due to the fact that older children are sent to hospitals for adults, but it is possible that the onset of second dentition may be a predisposing cause. In only one case was the child under two years of age, and I have not seen acute appendi-

citis in an infant under one year, but this is not the case in general peritonitis due to other causes. The cases were admitted to hospital and for the most part operated upon from the second to the fourth day, but 9 cases were operated on within the first twenty-four hours; 16 were received after the first week, and 8 after a fortnight's illness.

The cardinal symptoms of this condition in children vary little from those in the adult, but the site of the pain localized and indicated by the child when asked is a sign of the greatest importance. If the child places his hand over the right iliac fossa or umbilicus, and has at the same time fever and increase of the pulse rate, this alone is in my opinion highly significant of appendicitis, without further examination for local tenderness, etc.

The position of the appendix is of considerable importance, for several reasons.

1. In its influence on the course of the disease.
2. As regards the symptoms.
3. As regards localization of suppuration.

The appendix lies in nearly 33 per cent. of cases retrocæcally; out of 102 cases it was situated behind the cæcum in 32. In one case the tip of the appendix extended so high as to touch the neck of the gall-bladder. These cases of retrocæcal appendicitis come to the hands of the surgeon later than any other variety. The signs and symptoms are less pronounced and vomiting and diarrhoea are much less frequently present in such cases. In only one out of the 32 cases had the condition been present less than 4 days, and in most cases a localized abscess was present. General peritonitis in the retrocæcal position occurred only on one occasion, but in this case the appendix lay intraperitoneally on the inner side of the ascending colon, and the case terminated fatally.

From the operative stand-point they present greater difficulty than other cases, but the operation is as a rule simplified by dividing the base of the appendix in the first instance, and removing it afterwards to the tip. In two cases the abscess was perinephritic, and in one instance was sent to the hospital as a spinal abscess by the attending doctor. If the appendix perforates near its tip, when lying retrocæcally, the resulting abscess may simulate markedly a perinephritic abscess of cellulitic or renal origin. Two cases out of

32 died, one where a subphrenic abscess formed, and the other mentioned, of general peritonitis.

The next most frequent positions would appear to be in the iliac fossa in 21 cases. There is in many cases pelvic peritonitis, although the appendix itself does not lie over the brim of the pelvis. In one case the appendix was adherent to the common iliac and external iliac vessels, a state of matters which renders its removal difficult, and the risk of secondary hemorrhage considerable. A localized abscess was present in about half the cases, and in one was situated in the inguinal region, tending to pass into the inguinal canal. There were three deaths in the iliac variety. In 13 cases the appendix lay over the brim in the true pelvis. In these cases almost without exception the symptoms were referred to the pelvis, without marked rigidity of the abdomen. Irritation of the bladder shown by frequency of micturition, was a prominent sign and in two cases some diarrhoea and tenesmus, where the inflamed appendix lay alongside the rectum. In this variety there were two deaths.

Where the appendix lies directly inwards, the course of the disease for obvious anatomical reasons is more severe; the condition being almost invariably associated with general peritonitis. Eighteen cases had this position of the appendix, and of these 5 ended fatally, giving a mortality of 27 per cent. The prognosis is undoubtedly grave in this variety, as generalized peritonitis commencing above the pelvic region is usually present. They in some instances give rise to localized abscesses, when the inflammatory process is shut off by the omentum rolling itself around the appendix, and in these cases the abscess is usually situated behind the right rectus, and may even rupture internally at the umbilicus if let alone.

The position of the appendix therefore has a considerable influence on the prognosis and mortality, varying in the present series of cases from 8 per cent. in the retrocaecal variety to 27 per cent. where the appendix lies towards the general peritoneal cavity.

Males seem to be more liable to the condition than females, in proportion of 66 to 36, and the age of incidence is most frequent from the 7th to the 9th year. The youngest child was 21 months old.

## IMPROVED ŒSOPHAGEAL INSTRUMENTS

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THE accidental lodging of foreign bodies within the œsophagus is by no means uncommon, and in almost every case the intense distress suffered by the patient renders it imperative that immediate steps toward affording relief be taken. In many cases the question of affording relief for distress becomes secondary because it is not infrequent that the nature, conformation, and position of the articles lodged in the œsophagus are such as to endanger life. In all such instances the practitioner should if possible be provided with every means to enable him to secure the desired results conveniently and with dexterity. However, as with most accidents, the irony of fate usually decrees that when an individual is suffering from an œsophageal obstruction nobody in the vicinity is prepared with a set of instruments for removing the offending substance. In such cases the brain of the practitioner is taxed to produce results by resort to whatever methods his latent ingenuity may prompt. Desperation caused by facing a condition wherein the removal of a small substance is absolutely necessary if life is to be saved has very often spurred the mind to employ ways and means which are unique, to say the least.

An interesting experience illustrating such an instance may be cited as follows: While attending a patient on the second floor of a building, the first floor of which was occupied by a saloon, I was called downstairs to find a woman choking to death. She was blue in the face and almost exhausted. She lived in the neighborhood and while eating, a piece of spare-rib had lodged in the œsophagus with such results as to cause the woman to run to the saloon in the hope of securing help.

It was purely a coincidence that I was in that building at the time and naturally I was wholly unprepared for the case. However, something had to be done and that very quickly, and it struck

me that some fibrous foodstuff might be a desirable auxiliary in removing the obstruction, whereupon I asked for some raw sauerkraut. Telling the patient to swallow large quantities I called for a bottling-hose. The kraut did not pass the obstruction, whereupon I inserted the hose and thrusting downward I eventually succeeded in relieving the suffering woman. The kraut had evidently entangled itself about the bone, the effort of swallowing had the effect of forming a wad distending the œsophagus, and the pressure of the hose had dislodged the entire mass including the bone, thus saving the sufferer's life.

So far as I have been able to ascertain this "sauerkraut method" is original with me, and while it may not always prove efficacious it is at least worth trying when no better means are at hand. Some of the improvements illustrated herein owe their development to similar experiences, among them being the following exceedingly interesting case:

In the year 1905 a child was brought to me with the history that at the age of 18 months it had swallowed a silver "quarter," and that repeated efforts to remove same had been unsuccessful. The quarter had been lodged in the œsophagus at the trachial bifurcation, as shown by the X-ray, for a period of 220 days. In preparing to remove the coin I equipped myself with all available throat instruments usually employed for cases of the kind in hand and I felt after careful study of the possibilities that as a matter of diplomacy a further addition might prevent failure. Accordingly I made a careful inspection of the contents of my instrument cabinets and finally concluded to sacrifice an old horsehair probang for the desired purpose. After removing the horsehair I slotted the upper edge of the acorn tip (Fig. 1) in such manner that if I succeeded in passing the "quarter" the slots would, if they engaged the coin on the retractile movement, more than likely hold it fast and insure its removal. This extemporized adjunct, even though crude in construction, in actual fact proved itself superior to all the other instruments, for the reason that I only brought it into requisition after the others had failed, and when I did use it, it was after my colleagues had advised me that the best solution lay in forcing the coin down into the stomach as they concluded that its removal was practically impossible.

An ordinary stomach tube (Fig. 2) had its connection with a

case of extraordinary interest: A patient aged about 20 had been eating cold roast beef and bread and incidentally swallowed a right upper canine pivot tooth. Shortly after discovering the loss of the tooth she was seized with a severe pain located about eight inches

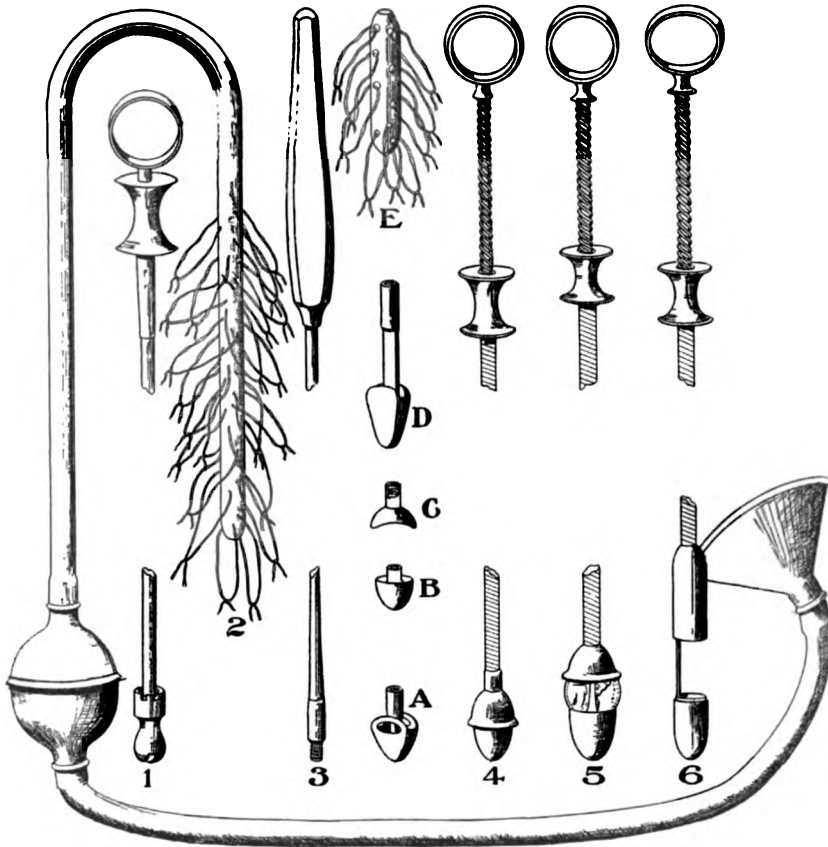


FIG. 1.—Improvised coin-catcher.  
 FIG. 2.—Snaring-net improvised from stomach-tube.  
 FIG. 3.—Stem for attaching the throat-buckets A, B, and D, depressor C, and metal loop-holder E.  
 FIGS. 4 and 5.—Throat-bucket with flanged spring cover.  
 FIG. 6.—Throat-bucket having a lateral stem and a spring cover not provided with a flange.

below the suprasternal notch. I was immediately summoned and arrived about 40 minutes after the swallowing of the pivot tooth. The patient explained that while the pain was at first mild but pressing in character it had rapidly become so severe as to make her feel faint. Examination showed the heart beating irregularly and spasmodically with great violence. The patient suffered chills

and spells of syncope while the pain about the heart increased continually, with shooting paroxysms travelling down the left arm. The fluctuating heart-action, the anxious countenance of the suffering girl, together with other symptoms, showed beyond peradventure that the tooth had found its way into the vicinity of the heart and that I was face to face with a situation which was at once serious and complex. The sharp pivot of the tooth had evidently penetrated the oesophageal wall, perforated the pericardium, and punctured the body of the heart, a condition of affairs which muscular action would probably tend to aggravate rather than to reduce. To use the usual instruments seemed to me to be attendant with too great a risk, since there existed a dangerous possibility that the introduction of hard-bodied extractors might intensify an already precarious condition by forcing the pivot deeper into the heart. My efforts to secure services of a specialist equipped with electrically lighted instruments, etc., proving unavailing, I was perforce compelled to devise some solution, since the condition of the patient with a pulse varying from 130 to 150 had become decidedly alarming.

Utilizing the stomach tube which I had in my case, I proceeded to thread it with numerous loops of strong silk as shown in Figure 2. These loops were about 4 cm. ( $1\frac{1}{2}$  inches) long, with knots outward to prevent the threads lying too close to the tube, thereby increasing the likelihood of securing a purchase on the offending tooth. I then gave the patient olive oil for lubricating purposes and twenty minutes after administering a hypodermic of morphine and atropine, I immersed the tube in the olive oil also and introduced it into the throat. The method proved successful, inasmuch as there was an immediate improvement in the patient's condition, the pain ceased at once and the heart action steadied. I had, however, erred in passing the tube down into the stomach, because it would appear that the loops engaged the tooth and had I made the retractile movement sooner than I did, I believe I would have succeeded in getting it out entirely, instead of dropping it into the stomach through the excess of motion downward. The patient had a temperature one to two degrees above normal for about one week, evidently due to the injury of the heart and pericardium, as the X-ray examination was negative. I am firmly convinced that the extemporized instrument saved the life of this girl.

A word of caution might not be amiss here regarding the use of rubber tubes for the purpose cited. It naturally follows that the tube should be sound, because under any other condition it might break in the retractile movement, thus complicating rather than remedying matters. There is no denying the fact that necessity is the mother of invention, and as one improvisation suggests another and another until a higher standard of practical perfection is attained, so in this matter of œsophageal instruments a varied experience has contributed materially toward the development of the means illustrated herein, a brief explanation of their intent being as follows:

Figs. 1 and 2 have already been referred to in the foregoing paragraphs. Fig. 3 is the handle and threaded stem for using attachments, *A*, *B*, *C*, *D*, and *E*.

*A* is a throat-bucket, oscillating on the stem, and similar to that already in use except that it is improved by having a threaded socket which is intended for screwing the instrument to the handle shown in Fig. 3. The advantage of this construction lies in the possibility of unscrewing the attachment in case it should get caught in such a manner as to make retraction dangerous or impossible, thus permitting the bucket to drop into the stomach.

*B* has an advantage over *A* in that its circular flange admits of engaging the body to be removed more readily, inasmuch as the point of purchase is continuous, and at the same time its acorn shape facilitates passing the object during insertion.

*C* is especially desirable for forcing down such impedimenta as cannot be removed otherwise. This attachment, in case of œsophageal ulcers, can be used handily as a curette.

*D* is more particularly an improvement on *A* in the matter of coin removing. While it has the throat-bucket form, it does not oscillate, and is therefore less liable to lacerate the œsophageal wall. This bucket is rigid on a flat piece of spring steel, mortised into the screw socket. This construction permits the engaged coin to lie flat against the stem and so facilitates removal.

*E* is a rounded cylindrical metal casting through which a number of perforations placed obliquely to each other have been drilled. The object of these perforations is to permit the attaching of silken loops, and the obliquity of the drilling insures a greater area of contiguity of the loops thus increasing the opportunity of engaging the



body to be removed, especially such articles as tacks, nails, pivot-teeth, etc.

Figs. 4 and 5 are a species of bucket shown respectively closed and open, and represent an improvement on Fig. 1, since it is often desirable after engaging a foreign body to clutch it. In the manipulation of this instrument, the flange of the upper half of the acorn-shaped tip will engage the article on the inserting movement, whereupon pressure on the handle will permit the forcing down of the lower half of the acorn, when slight lateral motion of the handle will permit the clutching of the body as the spring in handle is released. After grasping a coin by this method it is possible to throw the instrument a distance of 30 feet without dislodging the coin.

Fig. 6 is similar in its operative effect and manipulation, except that it does not have the flange as in Fig. 4 and is therefore easier to insert where small calibre is desired. Furthermore the stem is placed to one side instead of in the centre, thereby making it possible to close over articles when clutch is released. In the removal of small sharp articles this last advantage is particularly desirable, because if the operator can dislodge the obstruction and bring it within the cup, the release of the spring closes the cap and the retractile movement can in no way injure the œsophageal wall.

The principal object of the development of the several ideas shown in the illustrations was the providing of a set of œsophageal instruments which would be compact and at the same time be of practical use in almost any eventuality. Furthermore due care has been given to the necessity of having such instruments reasonably safe in the hands of the least skilful practitioner. The handles are all flexible, a circumstance which cannot be overestimated, since it contributes so much to the effectiveness of the instruments and reduction of the dangers of laceration.

It will be evident to the reader that the improvisation of the stomach tube (Fig. 2) formed the prototype of attachment *E*, the latter naturally being a great improvement for the reason that the neat flexible handle admits of greater latitude in operation. The instruments here described would seem to offer desirable auxiliaries to the means now at hand for the removal of almost any kind of foreign body, presenting to the practitioner a *multum in parvo* combination. The illustrations practically demonstrate their entire evolution.

**A REPORT OF THE SURGICAL CLINIC AT ST. MARY'S  
HOSPITAL OF ROCHESTER, MINNESOTA, FOR  
THE MONTH OF JULY, 1910**

**BY E. H. BECKMAN, M.D.**  
Junior Surgeon to St. Mary's Hospital

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Six hundred and fifty-eight patients were operated upon in this month. It would be neither interesting nor instructive to review all of these cases in a report of this kind, as many of them are of minor importance and would not be particularly instructive. However, most of the major cases and any of the minor ones of interest will be reviewed.

Cases of the same kind will be grouped together and those of especial interest discussed, and in some instances the case-histories will be given. In others some of the details of the operation will be mentioned in order that the reader may get a better idea of the case. Where autopsy findings throw light upon conditions, they also are mentioned.

Ether given by the open or drop method is the anæsthetic used in this clinic. Chloroform is used occasionally, but principally to continue the anæsthetic in patients operated upon about the mouth and face where it is not possible to keep the mouth and nose sufficiently covered to give ether.

**OPERATIONS UPON THE HEAD AND NECK**

*Craniotomy.*—There were two craniotomies, one of them a decompression operation in a boy nineteen years old for impending blindness due to a cerebellar tumor. He had had symptoms for one-and-a-half years. The other, a man forty-three years of age, had a depressed fracture of the skull in the left frontal region twelve years ago. He had been well until one week before he came for examination when he had had an epileptic attack lasting three hours. At operation a depressed fracture with several pieces of necrotic bone was found. It would be interesting to know how long the necrosis had been present.

*Carcinoma.*—There were ten patients operated upon for cancer of the tongue, lip and mouth. All were males. In all but two some form of chronic irritation appeared to be one of the etiological factors in the disease. Six of the ten were heavy smokers, two had bad teeth and one of them a poorly fitted plate, which seemed to be the cause of the irritation. The youngest patient was thirty years of age, the next oldest was forty, and the others ranged up to sixty-eight years.

In four of the patients the cancer was on the lower lip; one of them had been treated with caustics and one for syphilis. Another of the group had had the growth on the lip removed with paste two years previously, and the fourth had had the tumor excised three years before. Both of the latter came on account of enlarged glands in the neck which had been noticed for ten weeks and three months respectively. There was no recurrence of the original growth. Of the remaining six cases, in two the growth was on the tongue, one in the floor of the mouth and three in the alveolar process and cheek.

In each case, the content of the anterior triangles of the neck on both sides including the submaxillary-salivary glands were removed and the growth itself excised where it was on the lip, or it was removed with the actual cautery if it was on the tongue or in the mouth.

*Tuberculosis.*—There were seven patients operated upon for tuberculosis of the cervical glands. The youngest was sixteen, and the oldest thirty-nine years of age. Two were males and the remaining five females. In five, the enlarged glands were noticed after the following diseases,—cold, measles, whooping-cough, tonsillitis and la grippe. In one case the glands had been enlarged for fourteen years without suppuration. One patient was operated upon for a so-called mixed tumor of the parotid gland. He was fifty-seven years old and had had a tumor under the ear removed twenty-seven years ago which had been present ten years. The tumor recurred one year after the operation and had continued to grow very slowly up to the present time. He had a facial paralysis resulting either from the former operation or from pressure of the growth. There was a tumor the size of a small lemon in the lower portion of the parotid gland on the left side.

The tumor was removed and a block dissection of the left side of the neck made. The pathologists reported the tumor to be an endothelioma.

*Diverticulum of the Œsophagus.*—A female sixty-one years of age gave the following history: Eight years ago she began to notice throat trouble, solid food seemed to stick in her throat. She experienced no trouble with liquids, although ice-water had caused her severe pain for four or five years. She was gradually growing worse and for the past year she had eructations of mucus and ingested fluids. At times a lump which felt like a puff-ball appeared at the suprasternal notch. At the time of examination she had to drink fluids to swallow solid food. A small diverticulum of the œsophagus about one inch below the larynx was excised and the œsophagus sutured. The wound healed by primary union.

*Exophthalmic Goitre.*—There were twenty-two patients with exophthalmic goitre, two males and twenty females. Eight of these patients had a portion of the thyroid gland removed (usually the right lobe and isthmus); the other fourteen had both superior thyroid arteries and veins ligated. Operation of ligation of the superior thyroid arteries and veins is performed upon two classes of hyperthyroidism cases. First, beginning cases of a milder degree. It is hoped in these cases that the procedure will produce a cure. Second, more advanced severe cases or cases having a more or less acute hyperthyroidism at the time of operation. This simple operation is performed with the hope of relieving some of their hyperthyroidism and thus improving their condition so that a portion of the gland may be removed at a later time with safety.

Five of these patients had secondary hyperthyroidism having had the enlarged gland from two to sixteen years before symptoms of hyperthyroidism appeared. Four out of this group of five cases were females. The one male patient had a ligation performed, and a portion of the gland was removed later.

Four of the remaining seventeen patients had the right lobe and isthmus of the thyroid gland removed, one of them having had a ligation of the superior thyroid arteries performed at home six months before. The remaining thirteen had a ligation of both superior thyroid arteries and veins.

*Simple Goitre.*—Thirty patients, all females, were operated upon for simple goitre.

In sixteen, a partial thyroidectomy was performed. In fourteen cysts or adenomata were enucleated. One patient thirty-seven years of age had noticed a small tumor in the left lobe of the thyroid gland two years before which had steadily increased in size until at the time of examination it was the size of a small hen's egg. The tumor proved to be a carcinoma entirely confined to the left lobe of the gland.

*Breast.*—There were ten patients operated upon for carcinoma of the breast. All were married women, the youngest thirty-five and the oldest eighty-five years of age. The growths had been noticed from five weeks up to sixteen years. In three instances a history of injury was obtained; in one case fourteen years before, in another three years. One woman forty years old with a rapidly growing carcinoma of four months' duration, had had a suppurative mastitis with multiple incisions four years before. In two instances the carcinoma was a malignant degeneration of cysts. The growth in these cases had been present for two and seven years respectively. In one patient with a tumor of six weeks duration, the history showed that her mother had been operated ten years before for cancer of the breast which had not recurred, and that two aunts on her mother's side, had cancer of the breast.

In all of these cases the operation was a Halsted amputation, but the closure was by the flap method of Jabez Jackson. In addition to the above cases, there were seven women operated upon for benign tumors of the breast.

#### OPERATIONS UPON THE GALL-BLADDER AND DUCTS

*Cholecystectomies.*—There were eleven cholecystectomies. It is the custom in this clinic to remove the gall-bladder when it shows enough evidence of disease to destroy its function, or when stones are crowded into the cystic duct so firmly that their removal will permanently destroy the duct. There were stones in the gall-bladders of seven of these patients. In the remaining four there was so much evidence of total destruction of function of the gall-bladder as the result of cholecystitis that it was removed.

*Cholecystostomies.*—There were twenty patients on whom a cholecystostomy for stones was performed, and eight with cholecystitis without stones in whom the gall-bladder was drained. Seven

of these twenty-eight patients were males and twenty-one were females. In one patient there was a marked empyema of the gall-bladder at the time of operation, and in another a perforation had occurred from the gall-bladder into the duodenum.

*Common Duct.*—Seven patients were operated upon for stone in the common bile-duct, five females and two males. In two of the cases there were evidences of pancreatitis. In one case the cystic duct was dilated so much that the stones in the common duct could be removed through it without incising the common duct. One female, fifty-nine years of age had been operated upon here two years and three months before for stones in the common duct. The operation notes show that a probe was passed through the common duct into the duodenum at that time. For a year and ten months she appeared to be well, and then began to have symptoms of common duct obstruction which continued until her second operation. At this operation the common and hepatic ducts were filled with stones and stony material. This is, then, a case of reformation of stones in the common and hepatic ducts.

A woman sixty-one years of age was operated upon two years before in this clinic for a hernia of the small intestine through the mesocolic omentum, and for a large duodenal ulcer. The hernia was reduced, the tear in the mesocolon repaired and a posterior gastrojejunostomy performed at that time. She remained well until three months before her return, when she began to lose flesh and became extremely jaundiced. At operation a large tumor of the pancreas was discovered which was obstructing the common duct. The gall-bladder was enormously distended. A cholecystojejunostomy was performed. The patient died the day following. Autopsy showed the tumor to be a carcinoma of the pancreas.

#### OPERATIONS UPON THE STOMACH AND INTESTINES

*Ulcer of the Duodenum.*—There are seventeen patients operated upon for ulcer of the duodenum, four females and thirteen males. All of these patients gave histories of attacks of stomach trouble with intervals of freedom from symptoms. The average duration of the stomach symptoms was twelve and one-half years. A posterior gastrojejunostomy was performed in twelve cases. In seven the ulcer was covered with mattress sutures of linen in addition

to the gastrojejunostomy. Perforation had occurred in five of the cases. In two cases pyloroplasty was performed, also cholecystostomy, in one instance for stones and in the other for cholecystitis.

*Ulcer of the Stomach.*—There were three patients with ulcer of the stomach operated upon. In each case the ulcer was excised and the opening closed by a plastic operation. One of these patients had gall-stones which were removed.

*Cancer of the Stomach.*—There were three patients with cancer of the stomach in which a resection of the stomach was performed. Two others had carcinoma of the stomach, one at the cardia for which a gastrostomy was performed, in the other the carcinoma was found too extensive for removal. Another patient with symptoms of duodenal ulcer was found to have a moderate obstruction at the pylorus from adhesions of unknown origin. The adhesions were separated.

*Intestines.*—There were eight operations upon the intestines, four of these were resections, three for carcinoma and one for tuberculosis. One patient fifty-four years old had a carcinoma in the transverse colon which had produced symptoms for nine months, during which time he had lost thirty pounds in weight. Six inches of the colon were resected. Another male, forty years old had had symptoms for eight months with a tumor in the region of the cæcum for one month. He had lost fifty pounds in weight. A carcinoma of the cæcum was found, the cæcum and six inches of the ileum were resected, and a lateral anastomosis made. The third carcinoma was in a man seventy-three years old, who had had an appendectomy for acute appendicitis a year previously. He felt well for about one month, when he began to have pain in the right side not localized. Three months after the operation he had an acute obstruction for which an operation was performed. The operator found that adhesions from his appendectomy had caused the obstruction. After the second operation he had further attacks resembling obstruction, and had to keep the bowels loose to get a movement. There had been no blood in the stool. He lost twenty pounds in weight. The third operation revealed a carcinoma of the hepatic flexure and many adhesions about the cæcum from his former operations. The cæcum, ascending colon, and hepatic flexure along with eight inches of the ileum were resected and a

lateral anastomosis made between the ileum and transverse colon. The patient died of nephritis and bronchopneumonia.

The case of tuberculosis is interesting because an operation had been performed elsewhere three months before for suspected tuberculosis, but none was found and an appendectomy was done. The second operation revealed tuberculosis of a coil of the ileum near the cæcum. This loop, six inches in length, was resected.

A man sixty-two years of age had been struck in the right side of the abdomen with a pitch-fork handle nine months before. He was sick at that time for five weeks with a peritonitis and now suffered from tenderness in the abdomen, and attacks of obstruction. There was an indefinite thickening in the right iliac fossa. It was found that he had had a rupture of the ileum about eight inches from the cæcum which was closed by adhesions to the omentum and the wall of the abdomen. The opening was sutured, the appendix removed, and the extensive adhesions separated.

A colostomy was done in a baby one day old for imperforate anus. There were two carcinomas of the rectum operated upon, one in a man of fifty-four who had had symptoms for one and one-half years. A modified Kraske operation was performed. The other, a man sixty-one, had had trouble for five months. The Harrison-Cripps operation was performed.

*Herniotomies.*—There were thirty-seven patients with hernia operated upon.

Umbilical: One female was operated upon who had an umbilical hernia the size of a fist. She also had gall-stones which were removed.

Femoral: Two females and one male were operated upon for femoral hernia.

Ventral: One female with a postoperative ventral hernia following a pelvic abscess operated upon in this clinic three years before, was operated upon.

Inguinal: There were thirty-two males with inguinal hernia; ten double, one of them complicated with double undescended testicles. Of the remaining twenty-two, nine were on the left side and thirteen on the right. One patient had also an epigastric hernia which had recurred after two operations done elsewhere. Another patient had a large mass of paraffin lying near the internal



ring which had been injected nine months before for the purpose of curing hernia.

*Appendectomies.*—One hundred and seven patients were operated upon for disease of the appendix; forty-one for chronic appendicitis, or in the interval. Fecal concretions were found in ten of these forty-one appendices. Fifty patients were operated upon for acute or subacute appendicitis. In six instances there was an abscess outside the appendix. Ten of the entire number required drainage. In addition to these, thirty-seven appendices were removed secondarily in the course of other operations.

#### OPERATIONS UPON THE UTERUS AND ADNEXA

*Abdominal Hysterectomies.*—There were twenty-two women upon whom abdominal hysterectomy was performed. Twenty of them were married. Seventeen of the operations were performed on account of fibroid tumors in the uterus. Eight of the women had borne children, the other nine had never been pregnant. Three of the cases of fibroid were complicated by double infection in the tubes. Two others had cysts in one of the ovaries, and another of the patients had gall-stones which were removed. Of the five remaining cases, three had a severe infection in the pelvis which had involved the uterus. One had a carcinoma of the body of the uterus, and the other, a woman forty years of age had no children but had had many miscarriages and profuse menstruation for years. The flow had been so profuse for several months that she was extremely anæmic. She had a very thick bicornuate uterus with a much hypertrophied endometrium.

*Myomectomies.*—There were seven patients on whom an abdominal myomectomy was performed. In two of these there was a retroversion of the uterus which was corrected. In one, there was a dermoid cyst in one ovary, and in another, a double inflammatory condition of both tubes which necessitated their removal.

*Extra-uterine Pregnancy.*—Four patients were operated upon for ruptured tubal pregnancy. Three of them were married women and none of them had ever had children. Two of them had fibroids of the uterus; one so large that a hysterectomy was required. The fourth patient was a small undeveloped girl of fifteen years of age in whom menses had started one year before. She gave a history of moderate pain in the right inguinal fossa eight days

before, with nausea. The next day she felt better. Three days later she again had pain which lasted one hour. She vomited, was tender on the right side low down, and felt soreness on walking. There was no temperature. Twelve hours before coming for examination she again had pain lasting several hours. She was flowing at the time of examination and a mass could be felt low down in the pelvis on the right side. On account of the patient's age and childish appearance a tubal pregnancy was not suspected, and it was thought that she had an appendiceal abscess. Operation revealed many clots in the abdomen and a highly dilated unruptured Fallopian tube from which the blood was flowing.

*Prolapse of the Uterus.*—Seven patients were operated upon for prolapse of the uterus. A vaginal hysterectomy was performed in two cases, the Watkins-Wertheim operation was performed on three, and the remaining two patients had a high amputation of the cervix, an Alexander operation and a perineorrhaphy.

*Ovaries and Tubes.*—Twelve patients were operated upon for diseases of the ovaries or tubes or both. Four of these had double pyosalpinx. One inflammatory disease on one side only. One patient had a corpus-luteum cyst on the right side. Another had had a hysterectomy with the removal of one ovary three years before, she was operated upon for a parovarian cyst on the side of the remaining ovary. One patient had a pelvic abscess which drained through the vagina. The left ovary and tube, which were in the abscess cavity, were removed through the vaginal incision. Two patients were operated upon for adherent retroversion of the uterus. The Baldy-Webster method of shortening the round ligaments was used in each case. Another patient with a movable retroversion, had an Alexander operation, and the perineum repaired.

#### OPERATIONS UPON THE KIDNEY, BLADDER, AND PROSTATE

*Kidney.*—There were five nephrectomies for tuberculosis, one female and four males. In going over the histories one is surprised at their uniformity. The common symptom is frequency and bladder irritability with little or no colic or pain in the kidney. The patients were all between the ages of twenty-one and thirty. There were five nephrectomies for pyonephrosis with stones. These patients were from thirty-eight to fifty years of age. One of them

had had a stone removed from the same kidney fifteen months before elsewhere. One nephrectomy was performed in a woman seventy-two years of age for an infected hydronephrosis. There was one nephrectomy for traumatic rupture of the kidney. The patient, a man thirty-three years of age, had been crushed between the wheel and body of a carriage sixteen days previously. He had passed bloody urine, and two day before operation he had a chill and a temperature of 102° F. At operation many clots were found in the ruptured kidney. A girl sixteen years of age with a history of pain in the left kidney but no bladder symptoms, had a tumor which was noticed seventeen months previously. It had gradually grown until at the time of operation the mass filled the entire abdomen. The tumor (a sarcoma) was fully as large as a pregnant uterus at full term. A nephrectomy was performed and the patient made a good recovery.

*Prostate Gland and Bladder.*—There were ten prostatectomies. Nine were done through the perineum, and one, which had been drained six weeks before at his home with a suprapubic drain, was done by the suprapubic route. The youngest patient was sixty and the oldest seventy-five, average age sixty-five plus. One man sixty-seven years of age had had the middle lobe removed one and one-half years ago at his home, and had been compelled to use the catheter ever since. Another patient sixty years of age had had a prostatectomy done at home five years previously and now had partial obstruction from scar and remnants of remaining prostate. A third patient sixty-eight years of age had had a stone removed from the left kidney six years before. The kidney was now functioning normally. Two patients had carcinoma of the prostate, one, sixty-seven years of age had used a catheter entirely for three months, and the other, seventy-four years of age, continuously for one and a half years. This patient died of nephritis. In addition to these, three other patients had bladder operations. A man of forty-eight had a suprapubic cystostomy for stones. A patient sixty-two years of age had a suprapubic drain inserted for retention from an enlarged prostate. He has since had his prostate gland removed. Another patient sixty years of age, who had been passing blood for two years, was explored and found to have an extensive carcinoma of the bladder.

# **COLECTOMY FOR OBSTINATE CONSTIPATION; EXOPHTHALMIC GOITRE; APPENDICITIS; FIBROIDS; OVARIAN CYST; GALL-STONES; CERVICAL ULCERATION; PYLORIC OBSTRUCTION; VARICOSE VEINS \***

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## **EXCISION OF CÆCUM AND TRANSVERSE COLON FOR OBSTINATE CONSTIPATION**

**GENTLEMEN :—**This woman is twenty-two years of age, and ever since childhood she has been troubled with severe constipation. From the time she began to menstruate she has suffered from dysmenorrhœa, and during the past year she has had three or four attacks of appendicitis. The first thing we notice, after opening the abdomen, is that her transverse colon is prolapsed quite into the pelvis to the point which I show you. These webs of connective tissue which you see show that there has been a certain amount of inflammatory trouble, and it is very plain that it was almost impossible for her to have fecal material pass through the transverse colon. As the liquids contained in food are largely absorbed from the colon she has constantly absorbed the water or fluid that has been covering this fecal material, and as a result of it she is saturated with her own decomposing fecal material. This is a condition that Dr. Lane, of London, England, has described in a classical manner, and he finds that a number of these patients, especially women, are neurasthenic because they have never emptied their transverse colons. They are always carrying this enormous amount of septic material.

The method of treatment in cases of this kind consists in excising all of this prolapsed transverse colon. So far, I have simply removed the appendix in the usual manner because there is unmistakable evidence of chronic infection, and the question arises as to

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\* Surgical Clinic given at the Augustana Hospital.

whether I should remove the cæcum and transverse colon and implant the ileum into the sigmoid, or whether I should ignore the transverse colon. This operation Mr. Lane does usually in the milder cases. He makes the anastomosis between the sigmoid and the ileum and the transverse colon is left entirely out of the question because, after making the anastomosis between the transverse colon and ileum, the fecal circulation will take place through this short circuit and the chronic constipation will disappear. In order to allow a free escape for the fecal matter through the new ileocaecal valve, we make an anastomosis here of five centimetres (2 inches) in length. I make the same anastomosis that I do in doing a gastro-enterostomy. I place my double stomach forceps on the ileum and sigmoid, and put in a posterior row of Lembert sutures, which, as you see, will bring together the serous surfaces of the ileum and the sigmoid. I make my attachment five centimetres long (2 inches), so that there can be no doubt about the patient having a sufficient opening here. I have completed the posterior suture, and now, with a very sharp knife, I make an incision through the peritoneal and muscular coverings down to, but not through, the mucous covering. After this I begin with chromicized catgut at the opposite end, grasping all the layers and placing my sutures close enough to prevent hemorrhage. This suture, you see, grasps the peritoneum, muscle, and mucous membrane. I make this continuous suture with a straight needle and chromicized catgut, and now that I have reached the other end of this line which is to be sutured, I open the ileum and the colon, and carefully sponge away any of the contents that may leak out. There should be very little leakage as the intestines are clamped with stomach clamps. In order to prevent sliding back of the mucous membrane, I apply these mouse-tooth forceps, so that they will catch the mucous membrane, the muscle, and peritoneum, and now carefully sponge away what little discharge may come from the intestines. In order to remove the faeces accumulated in the colon, this patient received six ounces of castor oil in three doses, four hours apart, and several large enemas day before yesterday and no food, except broth by mouth.

We unite these edges by the use of the Connell suture. Bear in mind this little step in the use of the Connell suture which must be repeated constantly until the intestine is closed; you

must begin on the outside of the intestine, then pass the needle to the inside, grasping every layer, then passing it out at once from the inside to the outside, so that the little loop of your suture inverts the edge of the intestine when it is drawn tight, and you have no further concern about the line of suture because the Connell suture passes through all the layers and definitely controls the hemorrhage. Then it inverts the edge, and consequently definitely makes apposition of the serous surface.

Having finished the Connell suture we begin with the fine silk suture where we left it after making the posterior portion of it. Thus we place the Lembert suture over the entire surface which gives the Connell suture support, so that there cannot possibly be any giving way or leakage. I believe we can rely upon the Connell suture alone, but as it takes only two minutes to apply the Lembert suture over it, we shall do this and not take any chances.

In cases in which the pathological condition is still worse than in the one before us we perform the same operation with the addition of removing the entire colon to a point a little above the insertion of the ileum into the sigmoid flexure, thus preventing any possible accumulation of fæces in the now useless portion.

#### GOITRE

This young woman is twenty-one years of age. She is not engaged in any trade or profession, but lives at home. For three years she has observed a goitre. She also states that at the school which she attended almost all of the girls had goitres. We have operated in this clinic on many cases from the same village. Three months ago the patient noticed that she was becoming weaker, her hands began to tremble, and her heart to beat violently. During the last month or six weeks her eyes have become prominent. We find here a goitre about 12 cm. (5 inches) long, about 8 cm. (3 inches) wide, and 3 cm. (1 inch) thick. In order to secure a safe anæsthesia the patient is given one-fourth grain of morphine and one one-hundredth of a grain of atropine half an hour before the anæsthetic is given. She is very thoroughly anæsthetized with ether by the drop method and the field of operation is prepared. Just before beginning to operate we elevate the head of the operating table at an angle of forty-five degrees. The operation is done under

this original anæsthesia. The anæmia of the brain caused by this position makes it unnecessary to give more ether during the operation. I am going to reflect a flap in the shape of a horseshoe across the anterior surface of the neck, about 6 cm. ( $2\frac{1}{2}$  inches) above the sternum. I carry the platysma myoides and superficial fascia and skin back, so that you see the edge of the sternocleidomastoid muscle. The anterior surface of the goitre can be readily seen, and I am now separating the sternothyroid and sternohyoid muscles, as you see, on each side. Now, I expose the large lobe of the thyroid, and I find that it is possible for me, by means of a retractor, to draw to one side the sternocleidomastoid, and expose this lobe. I grasp the upper pole of the right lobe and with two pairs of Kocher forceps the thyroid artery and vein are clamped doubly. This gland is unusually long and hard, extending upward, making it difficult to reach. But, as you see, I have succeeded in grasping these vessels and am cutting between the forceps. I have reflected the capsule of the gland, and consequently I have no vessels to encounter until I approach the lower portion. At the upper end I find an enlarged communicating branch of the left superior thyroid vein. I grasp this vessel with forceps so that there will be no hemorrhage from that side. I must give my attention to the lower pole of this gland because of the inferior thyroid artery. At a point half way between the upper and the lower pole you find quite frequently the middle thyroid artery. I leave a portion of thyroid tissue half a centimetre ( $\frac{1}{5}$  inch) in diameter attached to the posterior capsule and trachea because by doing this there is no danger of injuring the parathyroid gland and recurrent laryngeal nerve. You practically never lose a case from anæsthesia if you do not give ether during the operation. If one continues giving ether while the operation progresses a little excess at a time when the patient is about exhausted nervously may be sufficient to prove fatal. I have seen four operations in which patients have died in that way. These patients are never very vigorous, and if you have them thoroughly under anæsthesia and hold them there, you may keep them so very nearly dead that the additional irritation of your operation may be just enough to kill them. On the other hand, if you get them thoroughly anæsthetized and then get the benefit of the anæmia of the brain, in addition to the anæsthesia they have already received, they will usually remain asleep throughout the operation. By giv-

ing these patients 0.015 Gm. ( $\frac{1}{4}$  grain) of morphine, and 0.001 Gm. ( $\frac{1}{100}$  grain) of atropine, half an hour before the operation, and then elevating the head at an angle of forty-five degrees, the patients having been thoroughly anæsthetized, they practically never wake up until the operation is completed.

We have removed the entire right lobe and isthmus and we have ligated a portion of the superior thyroid vein on the other side. A suture is also placed around the inferior thyroid vein on the left side. I will now grasp the superficial vein entering the left lobe and will ligate it separately at two points so as to cut off the blood supply to this lobe. I will not disturb the lobe itself, because I would much rather make a secondary operation than to make the primary operation too severe. A little secondary incision is made 2 cm. (0.8 inch) below the edge of the horseshoe-shaped incision, and dilated by means of a pair of forceps, and then a piece of formidin gauze is carried into this opening and the entire raw surface covered with it. Moreover, I take a small glass tube and pass it into the space from which I have removed this lobe so as to secure good drainage. Then the edges of the sternohyoid and sternothyroid muscles are sutured together again, leaving the anterior surface of the neck perfectly formed. Then we bring down the flap and take four catgut sutures through the fascia and the platysma myoides. Our reason for doing this is that there will be no tension whatever on the skin flap itself. There will be no pulling of this skin flap, and consequently the incision will leave no scar that can be seen at any distance. For a time we made a subcuticular suture throughout the incision, but that always leaves a thickened line.

The after-treatment in these cases is very important. I have seen a number of cases of exophthalmic goitre that have been operated on and had been made very much worse off after than before the operation, simply because it was supposed that after the operation was finished, the patient could do any foolish thing she wanted to. Such patients after these operations are generally the first ones to get up in the morning and the last ones to go to bed at night, and after a year or so they are surprised that they are just as bad or even worse than before they were operated on. These patients cannot take up extra work for instance in case of teachers for advancement in their profession simply because they have not the strength to do this with the condition of their hearts and nervous systems



which the hyperthyroidism has caused before the operation. One must insist upon the careful after-treatment of these cases, otherwise they will be handicapped all their lives. These patients should be taught the importance of not using coffee, tea, whisky, or tobacco.

I wish to direct your attention to a precaution which we take against the patient infecting her own wound with her breath. The mouth is thoroughly covered with a dozen thicknesses of gauze, held in place by an assistant who has nothing else to do, and it is wise to select a good assistant for that purpose because the position is a responsible one. If he drops the jaw the patient is liable partly to swallow the tongue, the gauze is liable to loosen, and the patient will very likely blow her breath directly into the wound.

#### APPENDICITIS

This patient has a very mild attack of appendicitis, and if she lived here in Chicago we would not advise immediate operation, but merely direct her diet and tell her to be very careful not to eat indigestible articles of food. But she lives in the pine forests in Florida; she had this slight attack of appendicitis down there, and consequently if she should have a severe attack at any time it would be rather dangerous, and it would be foolish for her to leave Chicago again with this obstructed and infected appendix. It is tied down by adhesions and full of pus at the present time because of a hard cicatricial contraction at the cæcal end.

We should never under any condition give a cathartic or food of any kind by mouth in an acute attack of appendicitis. Let us take some of the patients that have come to this hospital during this month. We have one that came in on the fifth day and one the seventh day with extensive peritonitis, and one has been here two weeks. They have had no cathartics, and will not be given any cathartics in the next month or two. In a certain proportion of cases a cathartic starts up peristalsis and kills the patients and in none of them is it needed, because by giving normal salt solution by rectum by the continuous drop method introduced by Murphy all of these cases will have spontaneous evacuation of the bowels in a few days, and if gastric lavage is employed the nausea and vomiting and gaseous distention will disappear very soon, provided neither food nor cathartics are given by mouth.

RECURRENT APPENDICITIS, COMPLICATED BY FIBROID TUMORS  
OF THE UTERUS

This patient is thirty-three years of age, married, and has two children. She gave birth to one dead child at term, and had one miscarriage at four and a half months. A year and a half ago she began to have mild attacks of appendicitis which recurred every few weeks.

Upon examination we find tenderness over McBurney's point; also tenderness in both tubes and tenderness over the gall-bladder. The tenderness over McBurney's point is attributed to the effects of the recurrent appendicitis. The distal end of the appendix is white and only one-half as thick as the proximal end, and it contains much connective tissue. Evidently she has had repeated attacks of appendicitis, and some connective tissue resulted from each attack, and this accounts for the obliteration of the distal end. We will remove the appendix. The pain in the region of the Fallopian tubes we attribute to the effects of the inflammatory trouble she had after her abortion. Now I have removed the appendix and will pack the intestines so as to have an opportunity to examine the pelvis. I find the uterus is studded with little pea-sized fibroid tumors. These we remove by clamping them off with forceps, because if we leave them in place they might enlarge and cause trouble. There is one fibroid the size of a large walnut in the left horn of the uterus. This we enucleate. She is only thirty-three years old, and if that tumor were left it would be likely to give her trouble. We carefully close the space left after the removal of this growth, using fine catgut. There are two things we are very careful about in doing this, one is to tie our sutures just tightly enough not to cause pressure necrosis, and the other one is to place one suture about 1 cm. (0.4 inch) beyond the distal end of the incision on each side. By making our sutures just tight enough to bring the surfaces together we avoid necrosis of the tissue of the uterus later on and guard against much annoyance, because usually when pressure necrosis takes place the patient develops temperature and is very ill for a time. This is true in cases in which the growth is so near the lining of the uterus that infection is likely to occur. The reason why we place the suture beyond the distal end of the

incision on each side is to overcome any hemorrhage from the blood-vessels that may enter the line of incision from these ends. Every now and then you will find that some blood oozes from these ends of the incision into the peritoneal cavity. By taking these precautions we have nothing to fear either from necrosis or from hemorrhage, and we leave the uterus in practically a normal condition.

Many of these patients have borne children after this operation. In a woman only thirty-three years of age this is an important matter.

Here I find I have torn an adhesion to this tube. The ovary was prolapsed behind the uterus and adherent there. The left tube is closed at the distal end because of adhesions to the ovary and broad ligament. I have loosened the adhesions to the broad ligament in bringing up the ovary and tube. This tube also shows the effects of inflammatory trouble, about  $1\frac{1}{2}$  cm. ( $\frac{1}{2}$  inch) from the uterine end. The distal end of the tube is distended with fluid, and this will account for the dysmenorrhœa. I shall now remove the ovary and tube, covering the stump which is left by suturing the median end of the round ligament directly over it with a few stitches of fine chromicized catgut which will prevent any of the other structures from becoming adherent at this point. We preserve the right ovary and tube. On the right side I will carry a little portion of the round ligament over the incision which I made in the right horn of the uterus in removing the fibroid. The fibroid was so situated in this case that it will not interfere with the lumen of the right Fallopian tube.

#### FIBROID TUMOR OF THE UTERUS COMPLICATED BY CYST OF THE OVARY AND GALL-STONES

Our next patient is a woman, forty years of age, who has a fibroid tumor of the uterus. She has never had any children. She has had considerable gastric disturbance, which we attribute partly to the obstruction caused by pressure of this fibroid upon the rectum and partly to the presence of gall-stones. She is very tender upon pressure over Mayo Robson's point, that is, a point half-way between the end of the ninth rib and the umbilicus. I also find a point of tenderness opposite the tenth rib posteriorly. Those two symptoms together, with chronic gastric disturbance, without pain upon press-

ure over the stomach, mean the presence of gall-stones. I find this patient also has much inflammatory trouble in her pelvis. The omentum is adherent to the anterior surface of the uterus and surface of the parietal peritoneum in front of the bladder. In many patients that condition is accompanied with gastric disturbance, because the omentum holds down the stomach. Furthermore, I find that the right ovary contains a cyst about the size of an orange, adherent to the posterior surface of the right broad ligament and laterally to the uterus. In loosening the adhesions I have ruptured the cyst. It is also adherent to the end of the fimbriated extremity of the right Fallopian tube which, as you see, is distended to the diameter of 2 cm. (0.8 inch) at this point. As I have said, I have ruptured the cyst, and will now remove the cyst and distended tube together. The uterus contains a small fibroid tumor, and it is universally adherent. In a woman at the age of forty the question arises as to whether it would be wise to loosen up all of these adhesions in the pelvis with the view of removing this uterus, or whether it would be better simply to remove the other ovary and tube also, and in that way deprive the uterus of its nourishment from the ovarian and infundibular arteries. It is doubtful which is the better course to pursue in a case of this kind because the tumor itself is not very large, and the amount of damage done in pulling it out is considerable. The chief advantage in removing the uterus, of course, will be that it will be no longer possible for the uterus to obstruct the rectum by its pressure. So I have loosened all of its adhesions, and I will now remove it. You see, the upper portion is one mass of fibrous tissue. Here is another small fibroid in the cervix the size of a hazelnut. We will close her abdominal cavity without removing the gall-stones because I think this is about enough for the patient to stand. We injure the tissues in a case like this more than ordinarily because of everything being so densely adherent, and when we consider that this woman is very fat, I think it would add more trauma to remove the gall-stones at this operation.

#### FIBROID TUMORS OF THE UTERUS

This patient is thirty-two years of age. She is suffering from multiple fibroid tumors of the uterus. She had a miscarriage last October, which was followed by infection and peritonitis, and now she has distention of both Fallopian tubes. She is extremely tender

upon pressure over the abdomen, and it is impossible to manipulate the tubes without anæsthetizing her. In fact, she is so tender that she could not bear the manipulations necessary for scrubbing her abdomen yesterday. This infection has existed since last October, practically ten months. She has received local treatment with very little or no benefit, and it is not at all likely that she will recover without an operation.

We will make quite a large incision in this case because we shall undoubtedly find the parts densely adherent. The appendix is tender, and here is an adhesion 2 cm. (0.8 inch) long between the ileum and the appendix which we clamp and cut. Here we have the right tube distended at the distal end, and closed at its fimbriated extremity, with adhesions to the posterior surface of the broad ligament and to the mesentery of the sigmoid. The left ovary is seemingly normal with the exception of some small adhesions. By beginning at the uterine end of the distended tube and advancing toward the distal end we can remove a pus tube without running the risk of rupturing it and spilling pus into the peritoneal cavity. Here we have a small fibroid tumor in the left broad ligament, and two further fibroids in the anterior portion of the uterus. We split the broad ligament, and enucleate this tumor, which is 3 cm. (1.2 inch) long, 2.5 cm. (1 inch) wide at its broadest portion, and 1.5 cm. (0.6 inch) wide at its narrowest portion. We enucleate this and close the incision in the broad ligament by means of continuous catgut sutures. We expose the other fibroids by making an incision down to the tumors and enucleating them. The right half of the uterus is normal. There is practically very little oozing from the surface from which I have removed these various tumors, but by lifting the uterus it is possible to control this oozing perfectly. Now all of these surfaces are covered with peritoneum by applying fine continuous catgut sutures, being careful to begin 1 cm. (0.4 inch) beyond one end of each incision, and completing the suture an equal distance beyond the other end. This leaves the uterus perfectly smooth and covered throughout with peritoneum, and all oozing is perfectly controlled.

Posteriorly, on the other side, we have a large fibroid tumor in the broad ligament. Over its upper surface you see a hard, distended, infected right Fallopian tube. This infection undoubtedly

took place at the time of the miscarriage, nine months ago. We will remove this ovary and tube, and then split the broad ligament transversely and enucleate the fibroid. You see how large these blood-vessels are that come into this tumor; so we grasp them separately. There is practically no hemorrhage from the pedicle. We have removed one large and six small fibroids from this patient, and we have left her entire uterus and one ovary. Menstruation will continue, but pregnancy will not occur. I pass this catgut suture around all of the vessels that supplied blood to this large fibroid so as to prevent hemorrhage; then I suture the split broad ligament in place, because if we neglect to do this the woman is likely to have a retroversion, and she would not be so well off as if we had removed the uterus down to the internal os. But when a patient is as young as this one, I believe the preservation of the uterus is the proper operation. This tumor extended over the posterior surface of the uterus, so that we shall have to cover that portion with peritoneum. Of course, it would have been simpler to have performed a hysterectomy here, but in a woman so young it is doubtful whether that would be a satisfactory operation from the stand-point of the patient. Now, we have all the fibroids and the right ovary and both tubes removed. The sigmoid flexure is now put in its proper place and the omentum brought down to cover up the small intestines, after which we close the abdominal wound.

## ULCERATION OF THE CERVIX UTERI

This is a very troublesome case of ulcerated cervix. The woman has a baby sixteen months old. She was severely lacerated at the time of delivery of the child, and has not been able to get relief from local treatment. I find that her cervix is very irregular. The anterior lip overhangs the posterior at least 2 cm. (0.8 inch), and there is a fissure to the right of the cervix and an ulcer on the posterior lip. The woman has been suffering severely from leucorrhœa all this time. Her uterus has not undergone involution properly, being at least four times the normal weight. By removing this hard cervical tissue containing this ulcer we hope to stimulate the uterus so that involution will take place. I have excised the posterior lip. Now, I apply these two sutures, which will unite the cervical mucous membrane with the mucous membrane of the

vaginal side of the cervix, and that will at once prevent atresia and also prevent ectropion later on, because the two mucous membranes meet to form a smooth union. I amputate this enlarged anterior lip which is hard and cicatricial. For quite a while we used ordinary catgut for this purpose, after having used chromicized suture material for years, but twice our sutures became absorbed early, and the patients nearly bled to death, and so since that time we have gone back to chromicized catgut sutures. I place two additional sutures, one on each side, grasping the tissues of the cervix, so that there will be no raw surface whatever. Now a suture is placed at the inner angle on each side, uniting the angle of the vaginal with the cervical mucous membrane, which further secures exact union there. I think it is quite worth while to adjust these sutures carefully, so that there will be no raw surface left. In other words, her cervix will be like that of a virgin. After operating upon the cervix we always place the uterus in its proper position, because sometimes the cervix is pulled down somewhat during the operation and the uterus may become displaced.

#### OBSTRUCTION OF THE PYLORUS

The next patient on whom we are to operate is fifty-two years of age. You will notice that she has a bad color of the skin everywhere. She is anæmic and emaciated. She vomits her food a couple of hours after she takes it. She has had gastric disturbance for a considerable time. She has pain upon pressure over Mayo Robson's point, which is half-way between the ninth rib and the umbilicus. At the present time her stomach reaches to a point 5 cm. (2 inches) above the umbilicus. She had two ounces of castor oil yesterday, and no food whatever since then, so that the stomach is not distended. Ordinarily it is distended to a point below the umbilicus. Over McBurney's point the right iliac fossa is 2 cm. (0.8 inch) higher than the left. Pressing upon this we feel a gurgling, and find a swelling which disappears temporarily. She carries her cæcum down in this iliac fossa, and it is full of gas. There is a painful spot half-way between the umbilicus and the lower end of the sternum. She has obstruction of the pylorus. She may also have gall-stones. Upon opening the abdomen by an incision splitting the upper portion of the right rectus abdominis

muscle longitudinally, you see the stomach presents itself at once, and although the stomach is not as greatly distended as it was, it is still distended. The gall-bladder also presents itself, and reaches down to a point below the umbilicus. Along the gastro-epiploic vessels there are lymph-nodes as large as lima beans. There is a strong broad adhesion between the inner surface of the liver, the gall-bladder, and the duodenum, which we have caught with hæmostatic forceps, cut, and ligated. Another adhesion extends the entire distance of the gall-bladder down to the cystic duct. This we also clamp and ligate. Her transverse colon is prolapsed to a point opposite the appendix. The cæcum is greatly distended, and the appendix is cicatricial throughout. We will remove the appendix. It is not likely at the present time it will cause much trouble, but being cicatricial it may obstruct and give rise to irritation. All of these adhesions undoubtedly are the cause of the obstruction to the cystic duct as well as the obstruction to the pyloric end of the stomach, and the question only remains whether the removal of these adhesions alone will relieve the obstruction. I am sure that the loosening of these adhesions would not in itself relieve the obstruction to the cystic duct, and so we will make an excision of the gall-bladder. This is accomplished by grasping the cystic duct with two pairs of hæmostatic forceps. In this case the cystic artery does not go with the cystic duct, but, as you notice, it takes an independent course just behind it. So I have had to grasp this separately with a pair of forceps. I place another pair of forceps on the neck of the gall-bladder and cut between these forceps and the neck, and then I simply peel out the gall-bladder, ligate the few adhesions, ligate the cystic artery separately, and then I leave one pair of forceps on the cystic duct. I could very easily ligate this, but I have found in cases in which I could not ligate the cystic duct conveniently, where I have left these forceps in place, the patients made fully as satisfactory a recovery as where I have been able to ligate it conveniently, and so in a very large number of cases I have regularly left the one pair of forceps in place. I place a gauze pad against the surface where I have removed the gall-bladder from the lower surface of the liver, as this will stop oozing very quickly.

The question arises as to whether this will relieve the obstruc-



tion sufficiently to cure the patient. I invaginate a portion of the stomach wall into the pylorus, pass my finger into the duodenum through the pylorus, my finger being covered with the stomach wall, then pass two fingers in there and stretch the pylorus, and I find that there still exists a little fissure which will have to be stretched. I think that will suffice. After removing this source of obstruction she will recover perhaps permanently. I place a cigarette drain down behind the liver into the kidney pouch on the outer side, another small cigarette drain on the inner side, and one more to the anterior side of the forceps, so that these cigarette drains thoroughly isolate the forceps that I leave attached to the cystic duct. Now, I close the abdominal cavity.

#### VARICOSE VEINS OF THE LEG

This patient has enormously enlarged varicose veins of the right leg, the short saphenous vein being involved. One of them is 2 cm. (0.8 inch) in diameter when he stands. At the lower third there is a little point where other veins form an aneurism about one centimetre in diameter which has so thin a wall that there is danger of rupture. We will excise the entire vein and ligate the communicating branch. The Esmarch constrictor will be applied after holding the leg in a vertical position so as to empty the veins and not cover the whole surface with blood while we are operating. Even after applying the Esmarch constrictor these veins contain quite a quantity of blood. We will cut off these veins everywhere so that they cannot possibly unite again. You will notice I am making little punctures or openings in about a dozen places to secure drainage of this large skin flap. We have excised the largest of these veins, and the others we have cut off at various points, so they cannot reunite and the return circulation must take place through the deep veins. Before we remove the Esmarch constrictor we apply a large dressing, and hold it in place with a soft broad gauze roller, so that the patient will not lose much blood from oozing. The leg will be kept in an elevated position for one week after the operation.

Before we send him home from the hospital we shall apply a cast composed of narrow gauze bandages saturated with Unna's paste, which contains the following ingredients: sheet gelatin, 4 parts; zinc oxide powder, 4 parts; glycerine, 10 parts; water, 10 parts.

## TRAUMATIC NEUROSES \*

BY ARTHUR DEAN BEVAN, M.D.

CHICAGO

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GENTLEMEN: I am very glad to have the opportunity of saying a few words on traumatic neuroses. It is a subject that concerns every railroad surgeon, as well as the officials of the roads themselves. It also concerns very much the claim agents, the attorneys, and even the Board of Directors, because very often these cases with their resulting costs interfere very much with the financial showing of the railroad. When I was a railroad surgeon I devoted a good deal of attention to it, and had a peculiarly favorable opportunity of studying some of these cases in an experimental way. My talk this afternoon will be perfectly informal and somewhat reminiscent.

A good many of you probably will remember that when we began to study medicine in 1880, and some of you a good deal later, that one of the interesting text-books of the day was a text-book on surgery by John Erichsen, who at that time was the most prominent English surgeon. His period of activity was during the years 1850 to 1880, and that period corresponds closely with the building of the main lines of railroad in Great Britain. Erichsen discovered a new disease, and that new disease was introduced by the railroad-building at that time; I do not know of any more interesting monograph or article on any pathological and clinical subject than Erichsen's discussion of what he called concussion of the spine, and which was shortly afterwards pretty generally named Erichsen's disease. I came upon the scene as a medical student in 1880, and studied Erichsen with interest, and in 1885 I was made chief surgeon of what is now the western end of the Union Pacific, and also did some railroad work for the Northern Pacific and Southern Pacific, with headquarters in Portland, Oregon. As Chief Surgeon

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\* Remarks made before the Association of Erie Railroad Surgeons, October 5, 1910.

I was responsible to the management for the decision in many cases of injury which were brought to us for settlement. I was literally full of the monograph of Erichsen. I believed fully what he had said about concussion of the spine. It was fairly generally accepted, and I remember one of the first settlements I ever made I made after talking with the President of the ——— and ——— Railroad, in advising a settlement of \$2500 in the case of a man who had received an injury in a wreck, and who was a typical case, to my mind, of Erichsen's disease. The President of the road was also the active man in the management at that time. I told the President that I believed this man was permanently injured; I was sure he could not recover, and I advised settlement for \$2500. On my statement the settlement was made, and within three months thereafter we found that this man was locked up in the penitentiary in ——— for forging a note and that immediately on receipt of the \$2500 he had recovered. I began to open my eyes. I read the literature very carefully and found the first real contradiction of Erichsen's theory was made by Page. Most of you have read this. It is rather reminiscent to bring up this phase of the subject. Page and other English surgeons, who had had a large experience, found that these people recovered after a settlement was made. Page wrote a good monograph controverting the position of Erichsen. About that time we had practically one of the best examples on one of the roads with which I was connected of an accident which resembled as closely an experimental research in concussion of the spine as could possibly have been planned or probably as has ever occurred. We had a train with 256 passengers on it, going about 25 to 30 miles an hour, over half a mile of trestle, when the engine plunged through the trestle at about the centre and the entire train fell down about 25 feet; not a single car turned over except the baggage car. The cars containing passengers all remained on the rails. The whole trestle settled like a pack of cards, so that these 256 people received essentially the same injury. It was about eight o'clock in the evening; nobody in bed; everybody standing in the car or sitting in their seats, so it was exactly as if 256 people dropped and struck on their buttocks or feet with the added momentum of the train going 25 miles an hour. If you can produce a better piece of experimental

evidence in this class of work I do not know of it. We had complete control of the situation. There were 133 people hurt. There were only six people killed, and seventy-five people were carried in stretchers that night from the wreck to the relief trains that came from both ends of the line. We had a sufficient corps who made a close study of the cases, who followed them out, and analyzed them with great care. This wreck cost the railroad a great deal of money. It took about six years to clean it up, and at the end of that time we had everything sponged off the slate and we could study the result. Of these 133 people, there were 80 claims of Erichsen's disease which were put down under the head of concussion of the spine. Of these 80 claims most were claims of serious lesion of the nervous system. You are all familiar with the old claim that a permanent injury of the nervous system is followed by degeneration of the brain and the cord with a multitude of symptoms, but of these eighty claims there were really but two of actual lesion of the nervous system. One was a fracture of the spine, which we settled for \$20,000. This poor fellow later died following an operation for the removal of a stone in the bladder. Many of these cases of fracture of the spine, as you know, developed stone in the bladder or kidney. The other case of actual nerve injury was an injury of the musculospiral, with fracture of the humerus. Of the other 80 cases, there was not a single one in which there was an organic lesion of the spine, of the brain or of the nerves; in other words, of any part of the nervous system, and yet these cases altogether cost the railroad several hundred thousand dollars in settlements. It was one of the most expensive wrecks, with the exception of the New-York-Central tunnel wreck, the cost of which I do not know, that we have had in this country.

In analyzing these cases, as we had the opportunity of doing, and as we did, we found we could divide the cases very distinctly into two groups, namely, those cases where we had the immediate control of the patients, that is, where either myself or one of my assistants had immediate control of the patient. The second group of cases was under the immediate control of some other practitioner who was not in harmony with the railroad and whose interests were rather those of magnifying the injuries of the patients.

As the result of a study of this wreck there were certain con-

clusions which I arrived at, and they were these: These cases of traumatic neuroses are cases in which there is no actual injury or the actual organic injury is comparatively slight; they are all cases in which the real condition is a mental impression of the patient, and that mental impression is worth studying. It is a composite thing. In the first place, it is an impression due to the injury, shock, and fright, and possibly some slight injury to the individual. In the second place, it is an impression of hope of recovering something for the injury. That is the second factor in this composite picture, the hope of obtaining some compensation for the injury. And the third impression is one of probably the greatest moment, namely, the impression made upon the victim of this shock by the attending physician who first sees the case. I analyzed that side of the case with interest, and I was forced to the conclusion that the most important factor in the development of these cases of traumatic neuroses was the attending medical man; that almost without exception, at least with very few exceptions, those patients who were the victims of traumatic neuroses could easily have been prevented from passing into that condition, or could have been cured in the early history of the cases if they had been in the hands of an intelligent, non-partisan physician who was simply looking at these cases from a purely scientific stand-point with the idea of obtaining the early recovery of the patients. There was case after case illustrating that point, one very forcibly so. One man in the wreck was a big, husky German brewer, who lived about two hundred miles from the scene of the wreck. I saw him that night. It is true, he was shaken up with the rest. He felt weak-kneed, and all the surroundings were such as to give that individual a very profound mental impression of that great disaster. But he got on the next relief train and was taken to his home two hundred miles away. The country the next morning was full of the news of this great wreck. His people and his wife and children met him at the station; they had telegraphed all over the country for him. There was great excitement. They bundled him up to the house, sent for a physician, and at first the patient assured them that he was not much hurt. His doctor said he must go to bed and keep quiet for awhile. He told him that it was in such cases as his that so-called concussion of the spine developed. The neighbors all

came around; they were sympathetic, and he was made a good deal of in the little town in which he lived. He was the man of the hour and was shown as one of the victims of that wreck. The doctor kept him in bed for a few days, and then sent for a lawyer, and then the question arose about damages in the case. There was another factor in this case, namely, this man had recently lost heavily financially. His little brewery had not done very well. He stayed in bed, and at the end of six weeks I examined him. He had hemorrhoids. He did not look very well. He was not feeling very well. He had difficulty in getting his bowels moved. I found that he had been taking a good deal of bromides and some morphine—in fact, considerable morphine, so that accounted very well for his constipated bowels and his rather bad condition. I looked him all over carefully, and he said he could not walk. I asked him why he could not walk; he replied “because my doctor says I have concussion of the spine. I must keep quiet.” I said to him, “you can walk and you are not injured, you are frightened,” and I picked him up in my arms, stood him upon his feet, and for the first time in six weeks I had him walking around the room. I put him back into bed again. I said to him, “you can walk,” and he replied with tears in his eyes, and a husky voice, “you tell me, doctor, I am not hurt at all, but my doctor says I am permanently injured. Who am I to believe in this case?”

The result was he was brought into court at the end of two or three months on a stretcher. His case was tried, and he got a verdict in his favor of \$10,000. A compromise was made, and the railroad company settled for \$5000 or \$6000. Within a short time he was chopping wood and apparently in perfect health.

Now, my impression of these traumatic neuroses is that the majority of them are not even that. They are simply cases of malingering, cases of out-and-out fraud. I have time and again seen fraudulent claims, cases that have gone into court with the idea of obtaining damages, always with medical men and attorneys bolstering up the fraudulent claims. These, I think, we can throw out of consideration entirely in the consideration of this subject. But there is a great group of cases of traumatic neuroses in which the patients are to a certain degree honest or absolutely honest. These patients are really in a bad condition from the mental im-

pressions that have been made upon them by the wreck, by their families, by their attending physicians, and especially by the fact that their cases are medicolegal and they have the hope of settlement. The moral to my mind is this—and I think I can speak now with some degree of non-partisanship because I am not at all a railroad surgeon, but I am rather one of those who believe that the railroads of this country need a pretty severe lesson: The railroads from the stand-point of money ought to pay more than they do for the accidents that they are responsible for. I wish they were compelled to pay ten times as much as they now do to the people who are injured in railroad accidents, because when we look at the situation and find that we are killing 10,000 people and injuring 90,000 more in this country annually, it makes us feel that something should be done to prevent this slaughter; because, as compared with the British and Continental railroads, our accidents are very, very much more frequent, and anything which will make the railroad management of this country more alive to the importance of saving human life and preventing injuries is a good thing, and damage suits and big damage recoveries are one of the factors which will urge them to greater caution. On the other hand, looking at the matter from a purely scientific point of view, these cases of traumatic neuroses, as a rule, are instances which can be prevented by intelligent medical management and the patients saved much suffering and the railroads saved much money. They are cases in which there should not be very much dispute between experts, but unfortunately, as our courts are constituted, and testimony presented, we find real medical experts are easily produced on either side of the case.

I was in a case not long ago in which two of the cleverest neurologists of this city testified in a case where a man had received a scalp wound. After my testimony in the case, which was that it was a purely mental condition, and a traumatic neurosis, these two experts said, yes, it was a traumatic neurosis, but a very serious matter. Was there any organic lesion? No. But the higher brain centres were involved, which was more important than an actual organic lesion of the brain, and as the result of this testimony the man received a verdict against the railroad company for \$20,000, so that we got experts on both sides of the case. But these cases

can be largely prevented from developing by intelligent medical management, and whenever I see one of these cases of traumatic neurosis, the first thing I want to do is to spot the doctor in the case. I want to see if he is an ignoramus, or if he is a partisan or a fraud, because you will find that doctors who are largely responsible for the development of these traumatic neuroses fall into one of these three categories. Of course, there are cases of traumatic neurosis in which this element does not enter at all, in which there is no element of compensation, and again patients of that unstable nervous make-up in which a slight mental impression will produce really serious results, often results that lead to serious mental states, even to insanity, and we must admit the possibility of these cases although they are rare. I have seen a number of them in which there was no medicolegal element at all. But, if you analyze these cases, you will find usually some motive in the way of desire for sympathy, but here again the important factor is almost always the doctor. As an illustration, I remember very distinctly the wife of a colleague who was hurt while in a carriage accident. The carriage ran into another vehicle and she bruised her knee slightly. The woman was somewhat frightened. She went on about her shopping. Her husband was unusually sympathetic and solicitous about her welfare. She telephoned him about the accident. He immediately went home, called in a surgeon, and this surgeon put a plaster-of-Paris cast on the limb. It proved to be purely a case of hysterical knee-joint; there was no medicolegal element to it. The woman wore the plaster cast for six weeks, and at the end of another twelve weeks she became a confirmed neurotic. Eventually, she was taken to an insane asylum. This was purely a case of traumatic neurosis in a very unstable individual. Such cases are difficult or impossible to cure. They are, however, the exception and are fortunately rare. The ordinary case of traumatic neurosis can almost always be prevented or cut short or cured by intelligent medical management.



# Neurology

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## HYPNOSIS, ITS PSYCHOLOGICAL INTERPRETATION AND ITS PRACTICAL USE IN THE DIAGNOSIS AND TREATMENT OF DISEASE \*

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I SHALL endeavor to demonstrate and interpret to you to-day hypnosis as we have been employing it for the purpose of diagnosis and therapy at the Vanderbilt Clinic during the last year and a half, and shall show you a series of patients who have been treated in this way. It may interest you to know that this is the first clinic on hypnosis held at this university except for one by Dr. Starr in 1892, and that old as psychotherapy is in practice—older by far than all other therapy—it has rarely been taught at the colleges, and you are probably among the first to receive instruction in the subject. This is far from saying, however, that we have been timely in engaging in this work, or are as yet by any means sufficiently energetic in treating it as a subject *per se*, or in launching it as a propaganda in defiance of the false “isms” and “ologies” ripening in our land. Abroad the work has been carried forward since the middle of the Nineteenth Century, or even before, and schools and traditions in psychotherapy have grown up and are to-day walking abreast with other therapeutic endeavors.

In no other branch of our science do we lag so far behind the continental schools as in this, and in none are we so in need of sane and rational leverage as here, for our people are among the most gullible (and hysterical) in the world and are easily led

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\* Based on a Clinical Lecture held in the Department of Neurology at the College of Physicians and Surgeons, March 17, 1910.

into strange and absurd beliefs by anything novel and specious, especially if their intelligence be flattered, or the proposed adjuvant be coated with theological veneer.<sup>1</sup> And so it has come about that we have in large measure forgotten actual scientific reasoning, and have allowed metaphysical pseudo-science and religio-metaphysical doctrines to sprout until they have become a power and

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<sup>1</sup>Let me refer to Christian Science (and the vaguer imitations sometimes spoken of as New Thought, etc.) and Emmanuelism, movements which have made incredible strides within a few years and have flooded us with a prodigious literature. In spite of the grain of truth they may contain, they must be condemned from our point of view as being utterly unscientific, irrational and dangerous. Then there is Dowieism! It is scarcely conceivable that in the light of the Twentieth Century an imposter setting himself up as a second Elijah should be heralded as such by some quarter of a million people and receive their wealth and allegiance. Schlatter, though quite a different type of man, was just beginning to have a following when he starved in the desert. With us also lies the credit of having given birth to spiritualism and of fostering its growth. When in 1844 Davis (the "Poughkeepsie Seer") proclaimed that he was visited in a trance by the spirits of Galen and Swedenborg and had great things to divulge to the world, and in 1848 it was proclaimed that the Fox sisters (of Hydesville, N. Y.) had established communication with the dead, these startling phenomena were taken up quite seriously everywhere, so that very soon supernatural "knocking" was heard throughout the country, and it is recorded that only a year or two after (about 1850) there were some hundred or more individuals in New York itself claiming mediumistic powers and nearly as many more in the neighborhood. And the knocking and table-tipping infection has constantly spread until, alas, it even entered high circles. Let me also recall to you the famous Madam Blavatsky and the founding of the Theosophical Society in New York in the seventies. The preposterous story of the Brotherhood of "Mahatmas" in Tibet and the transcendent and miraculous powers of this fraternity was accepted by thousands. Then the imposture of its founder was definitely proved—but theosophy just kept on and is supposed at present to have a very large following. At Loma in California there is an immense theosophical school and you have all heard of its "purple mother"! It is not entirely impossible that at some ripe moment there may be a swarming of its young disciples, and theosophy be the next religious fad forced upon us. Here, too, in reference to our hysterical emotionalism, I may allude to our mania for "revivalism" and prayer-meeting epidemics (no other country on the globe has just this sort of thing. For an interesting account see the chapter "American Mental Epidemics" in Sidis's "The Psychology of Suggestion"), our exaggerated athletic spirit, which has apotheosized baseball and football and crowded the academic from our schools (see Professor Gayley's "Idols of Education" a book it would do every American educator and student good to read), our Trilby, Klondike, prize-fight crazes and those delightful asininities, the Billiken and the Teddy Bear, the latter having certainly gone beyond the bounds of a fad with us.

a menace among us. The medical profession itself is much to blame for this, for it has partly countenanced the work of the healers, saying for instance, that "science," etc., is good for functional diseases or neurotic individuals, forgetting that such cults belie real science and set at naught all that Biology, Bacteriology, Pathology, etc., have taught us, while on the other hand (with a few individual exceptions) it has ignored psychology and psychological medicine,<sup>2</sup> and has looked upon the study of mental influence as dominant over the body, of "fantasy as a worker of wonders," as unorthodox and unworthy of professional inquiry. And even to-day, in spite of the work and fame of the School of Nancy and its distinguished disciples (Liebault, Bernheim, Forel, Wetterstrand, Vogt, Moll, Jong, Voisin, Berillon, etc.) besides that of such other European workers in the field of pathological psychology as Ribot, Richet, Binet, Pierre Janet, Schrenck-Notzing, Krafft-Ebing, Ziehen, Bechterew, etc. (the list of names is far too long to give it completely), and in our own country especially Prince, Putnam, and Sidis, that important branch of psychotherapy which we are discussing to-day,—eminently important aside from its own value because out of it grew the study and practice of psychotherapy as we know it to-day and through it was opened the study of psychopathology which has enabled Science to take new and promising surveys in what had hitherto been unfertile and unpropitious fields of work,—is either looked at with sinister connotation by the profession and referred to as a discreditable or even dangerous procedure, or, at the other extreme held to be so much nonsense and quackery.

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<sup>2</sup> Since this clinic was held Professor Schurman of Cornell University in an address on the relation of the University to the Medical School said among other things in speaking of the studies he would prescribe for the prospective medical student, "To these I would add a subject which I never see mentioned by advocates of either of the extreme positions I have already described, I mean the important subject of the study of the mind and more particularly those aspects of it which are embraced in the modern science of experimental psychology. Considering the close relation between mind and body and the dependence of some diseases upon mental conditions, I am often amazed that medical men fail so completely to realize the importance of the study of the sciences of mind as a part of the curriculum of the preliminary education they lay down for the student of medicine." (*Jour. Am. Med. Assoc.*, April 16, 1910.)

Let me turn to our cases and you may judge for yourself from what you shall see. Before proceeding further I shall put several patients into hypnotic sleep so that you may observe the condition induced before you. This may not be as spectacular as you may have been led to expect, but purposing in our work as conservatively as possible we have omitted all theatrical display and unnecessary effect, striving only to cure our patients or mitigate those symptoms for which they have sought our aid.

You will notice that I have used various methods, there mere fascination of the eye, as it is termed, here having the patient gaze into a crystal sphere (Braid's method), here using verbal suggestion, here command, here by stroking with my hands, here using several methods combined. It does not matter what method we use as long as we attain the condition itself. Nor does it matter how deeply the patient sleeps, so long as he does not fall into real sleep,<sup>3</sup> in which state mental communication between his mind and mine ceases, or as is said, we are no longer *en rapport*.


Now what is the condition in which you see these patients? What is hypnosis? I shall try to give you some insight into this romantic realm of the mental by paralleling analogies from the normal waking state with its phenomena, and elucidating both by means of psychological interpretation. I shall not be able to satisfy you entirely, for on this subject we are in most respects as ignorant as upon that of sleep itself.<sup>4</sup> To be sure, many theories have been advanced in explanation, psychological, physiological, histological (Heidenhain, Charcot, Liebault and Bernheim, Vogt and Forel, Dessior, Wundt, Cappie, Culerre, Papin, Grasset, Lipps, etc.), none of which, however, has proved satis-

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<sup>3</sup> As to depth of hypnosis many gradations have been made (Charcot 3, Liebault 6, Bernheim 9, Forel again 3, Krafft-Ebing, Moll, Hirschlaff, etc. 2). For our purpose we may speak of superficial and deep real hypnosis and a hypnoid condition often autosuggested. For therapeutic purposes the first condition is sufficient, while good results are often obtainable even in the last.

<sup>4</sup> So the various authors are also at odds as to whether hypnosis is real sleep or allied to sleep, or an entirely dissimilar state. That it is in no sense like sleep is the opinion of Krafft-Ebing, Mendel, Döllken, M. Hirsch, Hirschlaff, etc. That it is similar to sleep is held by Liebault, Bernheim, Vogt, Forel, Lehman, Wundt, etc. That deep hypnosis is real sleep but the milder forms are not is held by Kraepelin, Delbœuf, Moll, etc. I myself am of the first opinion. See Chapter V, Moll, "Der Hypnotismus," Berlin, 1907

factory. And yet these endeavors to explain, especially by analogous phenomena in the normal waking and sleeping states, have done much to make hypnosis psychologically intelligible and to divest it of its mysticism and the hypnotist of supposed occult and supernatural power. We know now that the phenomenon rests with the subject, not with the experimenter (a fact claimed by the Abbé Faria as early as 1814), and that its secret is based upon suggestion.



Let us say tentatively that we have in hypnosis a changed psychic condition in which a relative or absolute *dissociation* has been brought about by *suggestion*. Of course this must be effectual suggestion. And upon the ability of making suggestion effective, or, in better words, of achieving the acceptance of suggestion, lies the talent of the psychotherapist and the might of hypnotism. This, as already said, does not necessitate any supernatural endowment, though it does certain qualities of personality, as for instance, aptitude for coercion, and possibly that cogent something which on the one hand spells the imperative behind all persuasion and on the other motivates the allaying of fear, the easing of distress, the leveling out of life's demures into faith and hope. This something works in our hands by insight into human nature and with knowledge of the mind's associative dynamics. For behind suggestion stands the law of association.

And yet this does not explain all on the subject's side, for even in the normal waking state we have our inexplicable subjugations, we know how certain individuals sway others, how great statesmen, scholars, and scientists have been dominated by inferior individuals, a fact often incredible to us, how we ourselves are constantly being swayed knowingly or unknowingly and, if challenged for the cause, could give no reason why. Upon this fact Moll bases his third attribute among the five which he draws up as characteristics of our soul life, namely (I quote only the first four, as the fifth does not concern us here) (1) That man has naturally a certain inclination toward being influenced by others, and toward believing much without consciously coming to logical deductions; (2) that when one believes or expects a thing there results a tendency for the corresponding psychological and physio-

logical process to set in;<sup>5</sup> (3) that we not only have an innate inclination toward being influenced by others, but a proneness to being influenced by *certain* others; (4) that such influence is the greater having once been demonstrated—or again, that having once been influenced, the tendency of being influenced by the same individual becomes stronger. Without going into deeper analysis of these psychological facts, let us instance their truth by citing occurrences from everyday life: Let some one yawn and we yawn. Let some one at an assembly cough, and soon many are coughing. Let some one look up into the air and every one follows suit. See others drinking and you too grow thirsty (you know the popular phrase “making the mouth water”). See a faucet running and you soon feel the desire to urinate (a little trick many physicians make use of in their daily practice). How often does one feel an itching after having seen one of the tribe of pediculi, or bed-bugs,—“imagines he has caught it”! The very thought of it may cause itching. Close your eyes and sleep may supervene. Children smile if we smile, or may cry if we look askance. How easily they get a headache if they hear their elders speaking of one. In fact nervous parents invariably psychically infect their children. Again, you see the flag and you are stirred with a feeling of patriotism. A folk song or a picture may induce nostalgia. Sadness or gladness may overcome you on the suggestion of sadness or gladness in a person, play, or book. Finally, let several people tell a man he looks ill, and by and by he begins to feel so.

To bring the second attribute into consideration: The dyspeptic thinking he will have a pain after his meal, soon has it. The stutterer fearing to stutter does so. Those who are annoyed by inopportune blushing or flushing find themselves flushing as soon

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<sup>5</sup>The importance of this cannot be overestimated in psychotherapeutic work. To produce the conviction of the desired effect, no matter whether this is done in the waking or hypnotic state, is really the “open sesame” to our success. Dubois in his crisp and sturdy way has said, “The neurotic finds himself on the road to recovery directly he has the conviction of being cured; he is to be considered cured the moment he himself believes it” (*Les Psychoneuroses*, etc., Leçon xvii, p. 245). He adds, “this is the thought the physician must bear in mind if he wishes to heal his patient. But it is not sufficient to take this skeptically. He (the physician) must himself be convinced and must be capable of psychically infecting his patient with the same conviction.”

as they are afraid (and ashamed) that this may occur. A mother fearing her infant may wake up and cry, believes every moment she hears the crying. Let one anxiously awaiting someone's return, strain his ear in the silence of the night, and he will every few moments imagine he is hearing footsteps. The false sense-perception is corrected again and again, and yet again does he imagine he hears the sound. The apprehensive "look-out" in the fog easily sees outlines of ships before him, and the eager explorer in the ice of the north constantly sees illusive lands on the horizon. I remember a hazing act at a certain society where a man after being blindfolded was told he had to give up a little blood for the human ink used in keeping the secret records. His arm was cleansed and he was then made to believe that a minute incision was being made (this was done with a sharp cold lead pencil), that unfortunately the cut had injured a vessel, that the bleeding could not easily be stopped, etc. At the same time water was dripped into a basin, drop by drop as if blood were flowing. Then someone said in a scared way, "better call a doctor quickly, that's unfortunate,"—and the blindfolded initiate fell over in a dead faint. Through suggestion he has been made to become pale, have cold, clammy hands, have an interrupted heart action, brain anæmia and the resultant syncopal attack. Boerhaave relates a similar story, that a man condemned to death was handed over to him; that having blindfolded him, he went through the pretence of opening his veins (much as in the above hazing act) and that when the bandage was later removed he found the man dead. Give a patient a placebo and have him think it is a narcotic, and he sleeps. The thought of having taken the wrong medicine or a poison by mistake has frequently caused collapse; and you know how surgical patients have died in collapse from fear before the operation was begun. This "expectant attention" is the cause of many neurasthenic and hysterical symptoms (it is the cardinal element in the sexual-neurasthenic) and of many illusions and hallucinations (as noted above). It explains the apparitions in many a ghost story, by the way, and is a prominent factor in not a few of the famous imaginative tales of Poe, Hoffmann, Pushkin, Turgenev, Gautier and Maupassant.

Finally to take up the third attribute (and with it the fourth),

you all know how the mere presence of certain physicians helps or cures a patient (they feel better "as soon as they enter the office"), this especially so if it has happened before; how certain great men, leaders without, have at home been "under the boot" of their housekeepers or inferior wives; how certain teachers have greater power over pupils, certain lecturers almost a fascinating influence over students (Plato, Newman, Pater,—and as a recent example, Professor G. E. Woodbery); how certain clergymen can stir more intensely than others; how weak-brained individuals have been great politicians and leaders of men, or again a Joan of Arc an inspiration to valor, a Napoleon among generals electrifying an army. Again, friendship or love for certain individuals makes us wish to serve them, and lets us discern qualities, virtues or beauties, where they often enough do not exist.<sup>6</sup> In fact love, or even mere sexual attachment, easily enthalls. The sexual influence is astonishingly powerful and its peculiar puissance has eluded all analysis. You all know the part mistresses have played in the lives of Kings and the histories of countries,—and no less in the drama of private lives. This sexual attraction has been the chief theme of great operas, poems, novels, etc. It has become the *Leitmotiv* because of the tragedy involved, the dark peremptories the Fates here spin. (Let me refer to Prevost's "Manon Lescault," Dumas' (fils) "Camille" and "The Clemenceau Case," D'Annunzio's "Triumph of Death," George Sand's "Leone Leoni," the Countesse de Martel's (Gyp) "The Fairy," Strinberg's "Fräulein Julia," Kleist's "Kätchen von Heilbronn," Wagner's "Tristan und Isolde," France's "Thaïs," etc.) Krafft-Ebing<sup>7</sup> also alludes to this and points out how the will in such cases may be so under the influence of another as to make this problem of the greatest criminopsychological interest. He named this condition *Geschlechtschörigkeit* (sexual servility). A person under such influence may be induced to do the greatest criminal act at the mere bidding. (You all know the story of Mérimée's "Carmen.") Here too we might place the entire chapter of Masochism (although this

<sup>6</sup> Schopenhauer interprets this condition differently by assuming that the "interest of the species" blinds the personal interest. (See essay on the Metaphysics of Love.) This theory is psychologically untenable.

<sup>7</sup> *Psychopathia Sexualis*, pp. 151 and 396.



of course belongs entirely to the pathological). In all these sexual instances we cannot explain the influence of the one individual over the other. Mythology and mediæval tale have taken this into account, and the power of demons and gods, of love-philters, amulets, aphrodisial draughts and animal magnetism have all been drawn upon to explain it, nor must we forget the "elective" theory of Goethe's novel "*Die Wahlverwandschaften*." Again, special individual influence is wielded under such guise as we have learned to know as authoritative, as influential with heaven, or with the spirits or with strange natural phenomena, *e.g.*, of anyone having attained position, of the clergy, monks and nuns, of gypsies, sooth-sayers, of hunchbacks (see the many historic and literary instances in the novels of Scott and Dickens, and several instances in Shakespeare), and finally of so-called mesmerists and hypnotists, the presumed unlimited power of the latter being especially due to stage exhibitions.

Let me sum up the truth of the foregoing analogies by saying that suggestibility or "credivity," as Bernheim has called this human proneness to idea-influence, is a fundamental characteristic of our normal mental life, that it is common to all of us without exception,<sup>8</sup> that credulity and expectation, the tendency to believe much without making conscious logical deductions, are normal psychological phenomena, which give the clue to the exteriorizing and actualizing of imagination and the development of suggestibility.<sup>9</sup> Then, too, the degree to which we are susceptible varies, depending upon person and place, upon momentary mood, mental state, and the associations evoked. Fatigue, perturbation, passion, intoxicants, and especially passive emotion enhance our impressionability and strongly reinforce our susceptibility to suggestion, *viz.*, the twilight, the dark, the reminiscent

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<sup>8</sup> Bernheim pointed out, and Forel also states, that the refractory cases only appeared to be so, were so in fact only for the time being, and depended upon momentary conditions ("psychological relationship" between the subject and operator at the time being). Forel believes that probably all healthy individuals are hypnotizable, and Vogt goes so far as to say that not only are all healthy individuals hypnotizable, but somnambulizable—that is it is possible to put them into deep hypnosis.

<sup>9</sup> So Carlyle also wrote, "not our logical, measurative faculty, but our imaginative one is King over us," etc. (*Sartor Resartus*.)

mood, perfumes and music, silence and the evanescent environs of dream-states. In short, life does not dabble in realities alone—we are truly such stuff as dreams are made of—and every moment we are here in time is our consciousness swayed and modulated by the mingled real and mirage reflection of environment and idea.

This brings us to a closer scrutiny of *suggestion* itself, and in gauging it psychologically we come to clearer understanding of its action in connection with hypnosis. Much thought has been used up on this word and many have tried to define it. The conception is extremely subtle and elusive. If I submit an idea to the mind, or if our sense organs send up impressions and the mind accepts them, this is akin to conviction, but not suggestion. A suggestion is really not the idea but an intimation of the idea. It becomes an idea if accepted in consciousness. I might say that in suggesting I place the "pictorial" of an idea before the mind, and the suggestion works inasmuch as consciousness proceeds in actualizing this as if it were an idea, in reacting as if what is only a "ghost" of the actual were in fact real. For example, if I place two electrodes to your body and say that now you feel a weak current, and if I really let some current go through and you do perceive it, this has not been suggestion. But if I say the current is now passing and you believe you feel it in spite of the fact that I am not letting any current pass, then I have *suggested* the current to you.<sup>10</sup> The suggestion has entered your mind, your mind took for actual what was not actual. Or I might say your imagination "exteriorized" the consciousness of the sensation of electricity, making real what was only mental. It was Lippe who in his fine analytical way pointed out that the psychological phenomenon, the psychophysical activity occurring in suggestion must rest upon acceptance of ideas under "inadequate conditions" and not upon logical deduction. In other words, adequate means to the production of conviction might be grounds or reasons for so believing, while the suggested production of conviction would have such

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\* This experiment was undertaken by L. Schnyder, who found that over two-thirds of those tested perceived sensations, attributed to the electric current. See l'examen de la suggestibilité chez les nerveux. Arch. de psych. No. 12, 1904. Cited by Dubois.

grounds or reasons wanting. For example, if I told some one that I just heard through a third party that during his absence his house had taken fire, my words would produce real, motor, and emotive ideas in his mind. But this would contain no element of suggestion, for he would believe with full conviction, knowing how possible such an occurrence is, and not having any cause to discredit my words. But, if sitting in his own room, I should tell him the room was full of smoke and he believed it (as he would under hypnosis) this would be the acceptance of an idea under inadequate conditions, without ground or reason. We may therefore now formulate our definition of suggestion as a phenomenon in which under inadequate provision certain sense-perceptions are created or ideas generated, and the motor consequence precipitated by awaking the consciousness of the actual entrance of such contents in the stream of consciousness. We have already said that this occurs often enough in daily life, that we are constantly believing things without first logically reasoning them out. Nor do I again need to cite analogies from normal mental activity to substantiate this first of the five "characteristics" alluded to above, though it might be well to reiterate here that there is no one who believes only what he has deduced from logical reasoning. In this connection Moll has said, that were we to go through such weighing and surveying of every sense-perception as it presents itself to consciousness, we would be eternally at this and never at all come to action.<sup>11</sup>

I have said that "credivity" or suggestibility is a normal psychological phenomenon, common to all of us and dominating daily life. There are, of course, bounds to normal suggestibility, although the normal is not sharply defined from the abnormal. Abnormally we see it in the hypersuggestibility of hysteria and hypnosis. In hypnosis therefore the normal suggestibility of an individual may be abnormally increased. Hence suggestion not accepted in the waking state (waking suggestion) may be accepted under hypnosis. In analyzing this greater suggestibility and

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<sup>11</sup> This seems to echo the dictum of Cardinal Newman, who wrote in reference to faith however (*Discussions and Arguments*, Ed. 1888, p. 295), "Life is for action. If we insist on proofs for everything, we shall never come to action: to act you must assume, and that assumption is faith."

credulity in the hypnotic state, we must look at suggestion from still another point of view, namely, the mind's acceptance of an association of ideas by the inhibition of the rest of consciousness, or by the *dissociation* of the mind's bulk of associated ideas which it has accumulated during its life experience. From this viewpoint therefore we see that upon the inability to associate with one's entire mind rest the condition of suggestibility or the acceptance of ideas under inadequate conditions.

But why will the mind believe under such inadequate conditions? Here we come to the crux of our question—the phenomenon of dissociation, the careful consideration of which had best be preceded by a brief review of the psychological elements in the thought-process itself. A stimulus to a sensory end-organ produces a neural excitation which is transmitted by way of the centripetal nerves to the brain. The excitation in the cortex awakens a conscious process which we call perception or sensation. We perceive as long as the stimulus acts. Directly the stimulus is no more, directly the excitation ceases, perception ceases and the conscious state as such completely vanishes. Yet a trace of the physiological process remains behind as a permanent alteration of the cells excited, possibly an alteration in the disposition of the molecules or a metabolic generation of a kind of residuum. The cells are thereafter predisposed to the same excitation, and should such again come to them, this former “presentation” again becomes conscious.<sup>12</sup> This alteration in the cells, this residual something, is memory.

But different excitations may be transmitted to the brain at the same time (as of the smell, touch, or sight of a rose for instance) and be simultaneously registered in the cortex. Now, anatomically the ganglion cells of the brain are connected and interconnected by nerve filaments. And one might conceive of the “residuum” overflowing the cell out of all sides into numerous nerve fibres, and flowing farthest into such as were also vibrating from the other end at which a ganglion cell had also been excited, and in this way, by some means as yet unknown to us, establishing paths

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<sup>12</sup> This latent “charge” or “potential” in the cell once excited has also been spoken of as the “unconscious presentation” which when excited again becomes *conscious*. See also next note.

of communication. These then are "predisposed" paths, avenues of least resistance when such excitement again suffuses the cell at either end. Now as the stimuli acting upon us are manifold, the excitation transmitted to the brain multiple and complex, the communications between ganglion cells are constantly legion. Therefore let any one cell (or, rather, group of cells, for in the brain it is not so simple) be stimulated, and hundreds of the cells near and far in the cortex will be consorted and set vibrating; a hundred memories become conscious. Hence, the association of ideas, the interconnected contents in consciousness.

Though hosts of cells are constantly operating in the stream of consciousness, the greater bulk of the cortical "residual" elements are inactive.<sup>13</sup> These elements, these slumbering ideas may be thought of as latent and potential in the brain, as in the suburbs and beyond the outskirts of our present thoughts, or as lying in far distant fields of memory. Yet there they are, our ideas of to-day, of yesterday, of many years ago, easily again awakened, easily again set pulsing, easily again drawn into the focus point of consciousness by a neural charge, coming down some narrow alley of association. Always is idea conjoined with idea, memory linked to associated memory. And in this way amid this multiplicity of unconscious and conscious presentations there exists a path-way system in the brain, and in the mental life an integral harmony.

And so we say that the mind is made up of a myriad of distinct ideas or memories affiliated into a concordance or unity. And this aggregate, this totality of memory-pictures stored in

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"*Der ganze Reichtum von Erfahrung den wir als das Ich in uns tragen*" writes Stadelmann, "*ist latent; nur von Zeit zu Zeit tritt aus diesem Vorrat etwas heraus ins Bewusstsein und dient uns zum Denken, zum Urteilen, zum Kritik üben.*"

Plato also makes mention of this phenomenon in his Dialogue of Theætetus, in which he says "Our soul (mind) is like an aviary, full of wild birds, flying all about the place, singly and in groups. You may possess them, but you have none in hand; and until you collect, comprehend and grasp your winged thoughts, you cannot be said to have them either." See also Th. Ziehen's "*Das Gedächtnis*," Berlin, 1908, p. 3-4, 28, and 30-31.

It may be kept in mind that the same is true of emotions as of ideas; the stream of consciousness at any moment is imbued with but a minute quantum of that slumbering within us. Alfred de Musset has expressed this in the familiar line "*Et l'on songe à tout ce qu'on aime, sans le savoir.*"

the cortex is the Ego, or spiritual-I.<sup>14</sup> Or again, we may say, the Ego is composed of all the psychological experiences that the mind has gathered and recorded. It stands as criterion of all life values. Each new perception as it enters consciousness is "sized up" by it, to which as a new content it now becomes a part. (So also in the sense of Herbart's "Apperception-mass.")

Should a content or association-complex become separated from this aggregate-mass or Ego, we have a dissociation,<sup>15</sup> in contradistinction to association. Certain single contents may be dissociated from the Ego or larger association complexes, in which latter case the integrity of our mind life may be cleft and double personality result. To a certain extent it is a natural normal phenomenon, as we shall see presently; abnormally it exists in hysteria, which disease is characterized by its great proneness to dissociations. It appears abnormally in the hypnotic state also. Its study leads us through by-paths of scientific research into the borderland states of mental vivacity and vagaries of the mind and coaxes away from the fundamentals into the overtones of soul life. Here we happen upon the problems of penumbral moods, the tenuity of the subemotional, the realms of dreams, the interdreamed waking states and the somnambulistic hallucinations, trances, the so-called subconscious or subliminal self and the strange alterations in personality.<sup>16</sup>

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<sup>14</sup> "Meines geistigen Ichs, das heist, eine Gesamtvorstellung aller der Erinnerungsbilder welcher in meiner Hirnrinde vorhanden sind" (Ziehen). See also Ziehen's *Leitfaden der Psych.*, etc., p. 213; also this author's "Erkenntnistheorie" chapter 9, p. 40.

<sup>15</sup> We are speaking here of psychic and not organic dissociation. The organic, for instance, occurs in aphasia after apoplexy, etc., and in the psychoses "with defect." A psychic, functional dissociation, however, may also occur in the psychoses. See Ziehen's *Psychiatrie*, p. 82., Stadelmann's "Aerztlich-pädagogische Vorschule," chapter xix. See especially the interesting and instructive case quoted by Taine (*De l'Intelligence*, 3rd edition, vol. ii, note p. 461). Also, cited by James (*Psychology*, vol. i, p. 377).

<sup>16</sup> See Janet's "The Major Symptoms of Hysteria"; Sidis's "The Psychology of Suggestion"; F. W. H. Meyers's "Human Personality, etc." (edit. 1907); Morton Prince's "The Dissociation of a Personality"; Alfred Binet's "La Suggestibilité" and "Alterations in Personality" (1896); Raymond et Janet's "Les Obsessions et la Psychasthénie"; Waldstein's "The Subconscious Self"; E. v. Hartmann's "The Philosophy of the Unconscious"; Jastrow's "On the Trail of the Subconscious"; Sidis and Goodhart's "Multiple Personality"; Janet's "L'Automatisme psychologique," Paris, 1889; M.

Let us here take up some ordinary examples of dissociation in daily life: We cannot remember something, we cannot at the moment bring it into consciousness. Yet it is registered in our mind. At some other time "it will come to us." It is for the moment dissociated from the Ego. Should an idea however become dissociated and not again find its way back to the aggregate association mass, we have absolute forgetting. So, too, we see a face that is most familiar, but for the moment "cannot place it." In our mind then we go through the curious process of making these familiar features fit in somewhere, and finally succeed; or without thinking further of the person suddenly it occurs to us when and where we have seen the face before, *i.e.*, the gap in the association train has been spanned. This may happen even in the case of a person whom we know well. It has occurred to all of us that we have passed an acquaintance without recognition and that a few minutes after we remember and say, "why that was so-and-so!" We know now who it was (and strangely too, we recollect now that we passed someone whom we knew) but at the moment of passing the memory of the individual was dissociated. Let a boy be deeply engrossed in his dime novel and he neither hears nor sees what is going on about him. He is "dead to the world" we say. Save for the story in hand, all is dissociated from his consciousness. Impressions certainly do reach his brain through the ears and eyes and by way of tactile sense, but these are not apperceived, not associated to the Ego. They may be registered reflexly (subconsciously) or they may not find anchorage and thus be forgotten.

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Dessoir's "Das Doppel-Ich," Leipzig, 1896; Schrenck-Notzing's "Ueber Spaltung der Persönlichkeit," Wien, 1896. Binet, "On Double Consciousness" (Eng. Trans.), 1889; Ribot's "Les maladies de la personnalité"; Dana in "Psychol. Review, 1894, p. 570; MacNish's Philosophy of Sleep (1831); Azam "Les alterations de la personnalité"; Revue Scientifique, 1883, 11, also his "Hypnotisme et double conscience, 1893; S. Weir Mitchell in The Transactions of the College of Phys. of Phila., March 4, 1888; Krafft-Ebing's "Hypnotische Experimente (Stuttgart, 1893). For "Dämmerzustände" see also W. R. Gower's "Borderline States of Epilepsy"; Raecke's "Die Transitorischen Bewusstseinsstörungen der Epileptiker" (Halle 1903). For such conditions in connection with the child, Ziehen's "Die Geisteskrankheiten des Kindesalters," Zweites Heft, Berlin, 1904. See also in Oppenheim's Lehrbuch, under Hysteria. Also Griesinger and Krafft-Ebing's Psychiatrie. A series of very interesting cases are described by R. Henneberg in "Spiritismus und Geistesstörung," Berlin, 1902.

Dissociation also occurs when we are tired or excited, at which time we may "forget ourselves" (associations of social etiquette, etc., become dissociated) or make "slips of the tongue" or "stick" in our speech. How often do we in this way lose the thread of the conversation, and becoming embarrassed, soliloquize "what was I saying!" The condition which we call "absent-mindedness" likewise belongs here. A curious dissociation also occurs in that condition in which we feel as if we have experienced a certain occurrence before, or when we have the strange sensation as if we were living some former experience over again. Here the occurrence is momentarily forgotten (dissociated) and at the next instant again perceived; it now appears familiar but is not associated in memory (Stadelmann). The time-span may appear long even if the dissociation was but momentary. This is a constant occurrence in dreams. For another interpretation see James, vol. i, p. 675-676.

Stadelmann in a study of the nervous child in the school, called attention to the fact that the pathologically tired child may make numerous mistakes in writing and talking through psychic dissociation. The letters drop out of words (and even letters out of other dissociated words may be placed instead), words out of sentences, or an idea out of a train of thought, so that the writing or speech of such a child appears "full of mistakes." This of course is of great importance to the pedagogue, who is to distinguish here between the lazy or backward child that does not do its work well and the neurotic or hysterical. Such dissociation Stadelmann noticed as a frequent forerunner of epilepsy, and as an early sign of mental disintegration. ("Das Nervenranke Kind in der Schule," *Montagsblatt der Magdeburgische Zeitung*, No. 22 and 23, 1906. Reprint from Faber, Magdeburg, 1907.)

Again, in a normal way, dissociation is a phenomenon in the process of mental evolution, for associations must be loosened and separated to allow other ideas to be linked in. In this way also, we "change our minds" or undergo a thorough upheaval or reconstruction of our beliefs, theories, etc. It is by dissociation too, that discrepancies occur in our memories of past events. Certain ideas drop out of the aggregate association mass and do not find their way back again. On the other hand imagined incidents



are interpolated. So it will be remembered that Goethe, writing his autobiography late in life, called it "*Aus meinem Leben, Wahrheit und Dichtung*,"<sup>17</sup> knowing that his memory did not hold absolutely true, that imagination had colored events and filled in the interstices, that many an incident in his life had been

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<sup>17</sup> The italics are mine. For an interesting commentary on this, see Goedeke's Preface, Stuttgart Edition, 1867, p. iii.

In this connection one may also note that mental condition of the "romantic phantasiist" or dreamer who soars much and comes down to earth but little, so also that of the visionary who "sees things" (as for instance the poet Blake, Swedenborg, and Newman). To the kinship between the "artistic temperament" and hysteria Möbius has called attention. Here dissociation plays a more or less important part in proportion to the extent of the abnormality of such minds. It is analogous to the hysterical (especially so of hysterical children), in whom one finds varying degrees of abnormal fabulizing and pathological lying ("pseudologia phantastica," Delbrück), which is to be attributed to transformation of memory-images (though the hysterical also lies consciously often enough, in order to make himself appear interesting). Such individuals have a "hypertrophied imagination." After they have heard or seen a thing, for instance, or even while they are looking on or listening their imagination paints in additions or alters the entire. On reproducing the memory picture, the imagined portion is accepted as real and becomes actual for them. (The same is frequently true of children who are psychopathological by heredity.) Sometimes the "lie" is outright and entirely fantastic; sometimes there is an intermingling of truth and fiction. Of course there is no dissociation in the case of lying for a purpose (to help out a friend, to shield oneself, etc.) nor where in mentally abnormal or other children the perceptions are dulled or power of attention weakened, or where the sharp outline of the memory image is lacking. (So, for instance in the very young. See W. Preyer's *Die Seele des Kindes*, p. 234). There are many stages from the normal child with heightened imaginative faculty, who still knows where reality ends and phantasy begins, through the condition in which one "lies so well that one believes it oneself," or in the case of the child who "makes believe" so intensely that he gets frightened (at the make-believe bear or Indian, etc.) up to the condition in which the mind fabulizes and knows no more what is true or false, or becomes even a prey to its own phantasy. The entire subject is important not only from the viewpoint of the physician but from that of the pedagogue. For literature, see Oppenheim, *Lehrbuch*, vol. ii, pp. 1049, 1051; Ziehen, *Psychiatrie*, pp. 563, 587; Bruns, "*Die Hysterie des Kindes*," p. 27, 28, and especially Groos, *Das Seelenleben des Kindes*, Chap. XII; Strohmayer, *Psychopathologie des Kindesalters*, Tübingen, 1910, pp. 42, 43, 97-101; Delbrück, *Die pathologische Lüge und die psychisch abnormen Schwindler*, Stuttgart, 1891; Kemsies "*Kinderlügen und Kinderaussagen*," *Zeit. f. paed. Psych. u. Path.*, vol. vii, viii, 1905, p. 183, etc.; Stanley Hall, *Adolescence*, vol. i, p. 349-353; Ribot's *L'Imagination criatrice*; Binswanger, *Die Hysterie*, Wien, 1904, s. 330-334.

"dreamed 'round" or even wholly "dreamed in." (So, for instance, that charming *Sesenheim Idyll*.)

Finally, sleep itself is dissociation (and what a mystery it has proved!) and in the dreams which come with sleep we have dissociation, for you all know how our wonted ethical, moral, and other associations here pale into forgetfulness and we enact the part of thief or pirate, murder in glee, or dally along a primrose path of rapine and outrage, all matter-of-fact and unconcerned. So again there may run a medley of utter absurdity through our minds,—a diffuse dissociation—a veritable jumbling up of memories and ideas.<sup>18</sup>

We have spoken of certain individuals being more suggestible than others. We may also say of these that their minds are more dissociable. An abnormal extreme of this I have already

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"The subject of dreams is not only highly interesting in connection with our subject (for we have many dream and inter-dreamed trance states to deal with) but is important for the purpose of diagnosis and therapy ("dream therapy"). The following facts may therefore be noted: O. Vogt speaks of two types of dreams (a) the ordinary, entirely dissociated, diffuse dreams, and (b) the more circumscribed dreams of the somnambulist which correspond to a narrowing in of the field of consciousness. Here the neural energy is as it were dammed up in a particular circuit. There occurs in this state of partial waking in the midst of general sleep. In this active field the clearness and keenness of perception are intensified. In this somnambulist state the ethical associations are usually (not always) maintained.

That even the most absurd dreams are only apparently so, and may be analyzed and rationally deciphered, and that dreams are even direct "keys" to the mysteries of subconscious cerebration is the theory of S. Freud. See "*Die Traumdeutung*," Leipzig, 1900, "*Über den Traum*," 1901 (in *Grenzfragen des nerven und Seelenlebens*). See also Stekel's "*Beiträge zur Traumdeutung*," *Jahrbuch Psychoanalytische und Psychopath. Forsch.*, 1907, p. 458.

Perez holds that the influence of dreams extends to our sentiments and our morality. The mental states which are produced with or without consciousness during sleep are the consequence of and the preparation for certain states of our waking hours. He believes that according to the nature of the dreams which it has during the night a child is more or less cheerful during the day, more or less inclined to be good or obedient (*The First Three Years of Childhood*, Eng. Trans., 1894). Ch. Lévêque has written similarly "It is possible that the cheerful or sad humors of the day are a faint repetition of the agitations experienced in sleep, and that all the workings of the mind during the night may help to produce certain actions of the day (cited by B. Perez).

The most thorough volume on dreams is that of Sante de Sanctis, "*I Sogni*" Turino, 1899 (Germ. trans. by Schmidt, Halle, 1907). See also the

said we find in hysteria. Forel<sup>19</sup> has defined this condition as a dissociative weakness of the brain which occasions a diseased auto-suggestibility with its tendency to functional disturbance of all sorts, etc. A similar exaggerated dissociability occurs in hypnosis. It is because of this that some (the Salpêtrière School) have called hypnosis an hysterical attack and claimed its occurrence only in the hysterical.<sup>20</sup> This however is surely not the case and is at present maintained only by certain followers of the Charcot tradition.

If it were true, then, as Forel has pointed out, some 95 or 96 per cent. of mortals (for so many are hypnotizable) are hysterical! Then too, a certain class of hysterical individuals, if these are to be accounted such, are not suggestible at all but strongly auto-suggestive. I have already spoken to you of and shown you these cases, on several occasions and have suggested for this class the name "wet-faced" or "moist-faced" type.<sup>21</sup> Such individuals

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following: Tissé, "Les rêves," Paris, 1890; Kronthal, "Der Schlaf des Andern" Halle, 1907; Moll's Hypnosis, 4th ed., p. 180-195. There is an interesting note on the dreams aroused by organic sensations in Ribot's "The Diseases of Personality" (Chap. I) and also mention of dreams forgotten during the day and returning distinct in all details on the moment of falling asleep on the following night, etc. (Chap. III). Many literary tales have been built upon this fact and one has become rather famous and is among the finest short stories to be found in any language, namely, Th. Gautier's "La Morte Amoureuse."

<sup>19</sup> Hypnotismus, 5th Edit., p. 150.

<sup>20</sup> Hirschlaff's brief summing up on this point is excellent. He says, "There is not the least doubt but that the superficial forms of hypnosis haven't the slightest, but absolutely not the slightest in common with hysteria. On the other hand deep somnambulism is in theoretical aspect related to hysteria inasmuch as both are probably due to the same fundamental neuro-psychic dissociation. . . . Yet the hypnotized is quiet and shows no spontaneous reactions; the hysterical in the usual attack shows spontaneous phenomena, clonic and tonic spasms, screaming, emotional outbreaks, etc." Hypnosis can also be induced in the non-hysterical alcoholic, etc., etc. See his book, p. 218-20. Also Forel, p. 151-2, and Loewenfeld (Der Hypnotismus, 1901) p. 85, et seq.

<sup>21</sup> In our clinical material these patients are strikingly similar. They have all been Poles, chiefly female, at or after puberty (I have seen no cases among children). They are almost always somewhat fleshy or slightly obese, their complexion ruddy, easily deepening to a flush, the arterioles in many cases seen to be dilated, the eyes wet, the entire face looking decidedly "moist," carrying an expression of painful self-sympathy. They cry easily and copiously. They also sweat profusely. They evidence every lack of energy,

can be hypnotized only with difficulty, if at all, and they prove refractory to every kind of treatment.

How is this extreme dissociability brought about in hypnosis? It is done by narrowing the range of consciousness. By having a subject concentrate his attention we eliminate all other impressions, exclude all other perceptions, and bring the brain's activity to a focus. This not only intensifies the perceptive activity in the contracted field of consciousness, but disengages it from the remaining association aggregate, which becomes relatively inactive, rests as it were, as the brain rests in sleep. It is dissociating special association complexes from the Ego, or speaking in different terms, it is inhibiting mental action. When we narrow the field of consciousness by concentrating the subject's attention we are inhibiting other fields of consciousness.

This does not occur in hypnosis alone; it will be remembered that hypnosis itself is brought about in the waking state. But normal cerebration, too, involves constant inhibition. To entertain an idea many other ideas must be suppressed. In fact in the process of thinking this inhibition is very necessary, otherwise our associations would commingle promiscuously and thought-confusion reign.

It is given into our hands therefore to force areas of the brain into activity and thus throw others into relative desuetude. This fortifies us with a strong psychic implement in our therapeutic armamentarium, for there occur in the act of cerebration two rather remarkable psychophysical effects, namely, *that in neural activity energy is discharged into motor or emotive action, unless such action be inhibited; and that such motor action or emotive effect inhibits the opposite or contrary issue.*

The comprehension of this is so significant for the actual character and will power. They complain of being abjectly miserable, of having pains *all over*, usually of a "burning" nature. The pain "wanders about" and at night keeps them from sleeping. They may also complain of paræsthesias, but pain is the cardinal symptom. Objective examination has usually been negative. These patients are mostly of an ignorant type, and appear insensate to all advice, suggestion, or logical persuasion. Psycho-analyses and association tests have proved impossible. The psychalgia is almost un-influencable by drug treatment. I have tried bromides, the coal-tar products and even large doses of codeine without effect. Nor has any other therapy seemed helpful in my hands.

manœuvring in the practice of psychotherapy that we must examine carefully the various elements in the psychological problem here involved. The elements in the complete neural charge and discharge occasioning a psychophysical process are excitation (external stimulus, afferent current, cortical reception) perception or sensation, association of ideas (or play of motives or deliberation), and finally action (or emotivity) or, *sensu stricto*, the resulting idea of motion precipitating the act, etc. (Ziehen). The neural afferent charge is therefore modified before it becomes efferently discharged. This modification occurs after the perception has worked in as an idea in association among the memory images left by former cortical excitations. It is milled as it were by the apperception mass and comes forth essentially altered. In this process another factor plays a very important rôle, namely, the positive or negative feeling or emotional tone (Ger., Gefühlston<sup>22</sup>). Not only is every perception accompanied by its emotional tone, but every memory has its pleasureable or displeasureable deposition, which is again actualized in the awakened memory.<sup>23</sup> These feelings are the motives to productivity. They become summated and discharged and so the impellents to action and deed. Every incoming nerve current to the perceptive centres therefore stirs up feeling, positive or negative, and in the association of ideas, in the engagement of motives there is a sort of battledoor and shuttlecock play—an associative grouping of memory images and sensations—and finally the positives or negatives (or neither) win; and this proves the *fiat* or *negat* upon which depends the discharge into the motor area and the consequent action. And herein also lies the secret of volition and *the psychology of will*.<sup>24</sup>

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<sup>22</sup> There is no English equivalent for this word. It has been translated as "emotional tone," "affective tone," "tone of feeling," etc., all of which terms however lack the finer nuance of the German. Frequently the word "interest" is used to express the same thought (so in James's work).

<sup>23</sup> For reference on this subject see Ziehen's "Psychology," 7th Edit., Chapter IX; also that author's monograph on Memory, Berlin, 1908, p. 14; also H. Stadelmann's *Aertzlich-pädagogische Vorschule*, etc., Chapter IV.

<sup>24</sup> "Das Wollen bezeichnet auch eine seelische Situation, welche ausschließlich durch ganz bestimmte Vorstellungen und Gefühlstöne gekennzeichnet ist." Ziehen, *loc. cit.*, p. 258, etc. "In dem Anstoss, den das Zentralnervensystem von aussen her durch den Reiz erhält, liegt schon im Keime der Impuls zur Ableitung den wir Wille genannt haben. . . . Wir müssen uns stets vor

Now let us momentarily return to the beginning of our former paragraph and schematize the neural process into three elements: (1) perception or sensation, (2) association of ideas or play of motives, and (3) action, etc. If we omit the first and third elements in this chain we have the psychical function of *thinking* (or reflection).<sup>25</sup> And yet this is but roughly true, for if we examine our problem carefully we find that the third element is really not entirely wanting in the process of thinking, and that in truth some energy is discharged into motor channels, and you will notice during this psychophysical activity that the forehead is furrowed, the jaw held somewhat firm, the lips moved or drawn tense, the neck musculature fitted in equipoise, and the entire body more or less "set." Again, thinking has been described as an abstinence from talking or acting,<sup>26</sup> in which conception is embraced the idea of a damming up of action (for talking is also action). In short, without piling up further citations, we may assume that every idea is suffused with a potential motor charge, or go even farther and say that more or less of the motor is actually always discharged.<sup>27</sup> Phylogenetically and ontogenetically, we may be reminded, motion and cognition go together, and so too in their areal projection on the cortex are topographically adjacent.<sup>28</sup>

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Augen halten, das wir unter Wille eine Summe von Vorgängen zu verstehen haben und nicht eine selbständig und unabhängig wirkende Kraft." H. Stadelmann, *loc. cit.*, p. 72. See also an excellent exposition of this conception in H. Münsterberg's "Psychotherapy," p. 90. (Will is also so treated by Herbart, Bain, Spencer, etc. Wundt, on the other hand, has come to quite contrary conclusions.) It is very important to keep this conception of will in mind, for it answers many of the questions asked of the hypnotist concerning the subjugation of will under hypnosis, the possible weakening of the will, etc. Will is only strengthened in the hands of the trained psychotherapist, never weakened. This is the conclusion that every careful specialist on the subject has come to. Pertinent to this see Aschaffenburg's remark on p. 796 of H. Curschmann's large recent compilation of nervous diseases, Berlin, 1909. See also p. 377, et. seq., Loewenfeld's *Hypnotismus*, 1901.

<sup>25</sup> Th. Ziehen's "Leitfaden der phys. Psych.," pp. 22, 212.

<sup>26</sup> A. Bain, *The Emotions and the Will*.

<sup>27</sup> Every impression which impinges on the incoming nerves produces some discharge down the outgoing ones, whether we be aware of it or not, etc. (James's "Psychology," vol. ii, p. 372). See also Ribot's "Psychol. of Attention," Chap. I, 2, and the following reference.

<sup>28</sup> M. Cohn, "Ueber das Denken," Berlin, 1910, p. 63, etc.

I have spoken of motion and emotion together. You will call to mind the existence of striped and unstriped muscle fibres, the voluntary and involuntary. Motivated action is associated with the former, the emotive (arteries, bladder, gastro-intestinal tract, etc.) chiefly with the latter. The latter too are especially the vegetative, automatic, unconscious processes. But both are intimately influenced and more or less swayed by cortical excitation. You know how one can often deduce the thoughts of an individual or the motives behind his thoughts by watching the action of his arteries and facial muscles (flushing, paling, stuttering, "expressions," etc.), and how because of this observance we say we "read people." Again, we are rather astonished when our thoughts are "read" as the muscular innervations which they occasion are accomplished unconsciously. Nor does this phenomenon occur only in the "mimic," though it is here that not only momentary thoughts are portrayed, but the larger bent of the soul-life is exquisitely graven and the lines of character indelibly etched in. It will be found, as Lange pointed out, that different thoughts, or rather optical memory-images will bring about unconscious movement in the eyes (think of the future and the eyes and lids are thrown somewhat upwards, think of the past, and the contrary occurs, etc.). Again, you know how the anal sphincter may contract in excitement, or the muscle fibres of the bladder, the latter causing constant micturition (as, for instance, in the case of the student awaiting an examination). Here too belong the unconscious rubbing of hands, wiggling of the fingers, tapping with the toes, picking at the nose, chewing the lips, scratching the head, being fidgety in general, and especially the incessant walking back and forth when agitated. (One may also attribute the agitation and great urgency to activity of the maniac to his "flight" of thoughts, and the slow movements, heaviness, inactivity, costiveness, and enuresis of the melancholic and hypochondriac to his slow state of mental activity.)

If we have said that the action may be inhibited, this does not imply that the excitation has not run into motor innervation. In such a case there may be a careful balancing of muscles giving

an equilibrium, an attitude, an attention,<sup>29</sup> or a general muscular "setting." Eventually therefore, the muscle is always at the end of the neural current,<sup>30</sup> and a motor element is the inevitable resultant in the arc of psychophysical activity.

As for the second effect referred to above, namely that motor action inhibits the opposite action, let us now word this somewhat differently and say that when the mind wills an action the opposite action is suppressed or inhibited. It appears that innervation simply blocks the motor path to the contrary innervation. You cannot inspire and expire or smile and frown at the same time, nor will to open and close the eyes simultaneously. To will the one necessitates the negation of the other. "There may be a wrangling between those two impulses," writes Münsterberg, "but as soon as my will stands for the one the other is really excluded. Any action which I am starting to do thus crowds out the impulse to the opposite action." How does this apply in the case of other than single ideas? Two separate ideas cannot be entertained at the same time, but simultaneous ideas blend into complex ideas. It is evident from this that the ideas must be closely associated and correlative. Only such ideas as "fit" into the same motor setting can merge and be thought of at the same time. We cannot ponder two themes at once. A content not harmonizing in the general motor poise, not congruent to the conceptual system is therefore dropped from the mind for the time being. You see of what great practical importance this phenomenon is to us as psychotherapists, for in wishing to banish a distressing train of thoughts or expel depression, we have but to suggest the opposed mental trend and we do so not only by constant and reiterated

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<sup>29</sup> The word attention is from the Latin "tendo," to stretch or strain; hence also our word "tension." Muscular "strain" is implied in the word, and such muscular strain or tension is always noticed when we "strain our attention," or "attend closely." This feeling is also present when we strive not to wink, not to smile, or "try to recollect," etc. The sensation emanates partly from tension in the tympanic membrane and the muscles used in accommodation and convergence, the frontalis, etc. See also James, *loc. cit.*, vol. i, p. 300-301, and Duchenne, cited by Ribot, *loc. cit.*, I, 2.

<sup>30</sup> It has even been held (J. Gerlach) that the muscle was nothing else but an expansion of the nerve. (See Cohn, *loc. cit.*, p. 68, etc.)



suggestion but by adding associations and thus strengthening the new premise.<sup>31</sup>

I have not dwelt upon the subject of attention, for it would take too long to discuss this important chapter in psychology. But what I have said of thinking applies here too, for attention is no more than a matter of ideation,—sensations or ideas are the motives occasioning attention, because they happen to be in the field at that moment by way of association, or because they are brought into focus by vividness, intensity, etc.,—and involves nothing *ultra* to the neural (psychophysical) process familiar to us.<sup>32</sup> When we will to attend a thing, it is willed here precisely

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<sup>31</sup> In cases of depression and melancholy hypnotic treatment proves most beneficial. I have seen several cases, which had been treated in very many ways and over a period of several years without success, begin to improve almost immediately under hypnotherapy. One endeavors in these cases to awaken positive emotional tones, a task not easily accomplished in the waking state, as such patients remember nothing pleasant, all happenings with which positive affective tones are connected seem to have dropped out of their memories. With the awakening of such positive feeling one also awakens hope in the patient, who having thought that he could never more experience joy, now begins to believe that probably after all he may be cured. This very hopefulness is the beginning of and the greatest adjuvant to his recovery.

In this connection it may also be pointed out that children unconsciously make use of the same phenomenon when they think of sexual things in order to banish fear at night. Inquiry into the causes and continuance of masturbation in young children and adults, has led me to believe that one, if not the chief reason, of continuance of this habit lies in the fact that the sexual being the very strongest of emotional tendencies, is brought into mind to crowd out the "being afraid" which especially nervous children and adults experience when alone in a room at night, or in the dark. Such thoughts are also used to dispel other worrying or distressing ideas in older people. In the treatment of manual or mental masturbation therefore, this should always be kept in mind.

Immanuel Kant in that now famous little volume "*Von der Macht des Gemüths*," also wrote how he was able to conquer his insomnia caused by severe spasms of pain, evidently due to gout, by bringing some other thought into his mind (not by expelling all thoughts—"thinking of nothing") and riveting his attention upon it—as for instance upon the name Cicero which evoked innumerable associations in his mind. Very soon the pain was forgotten and he would fall asleep (p. 26).

<sup>32</sup> "Apperception ist Eintritt einer Vorstellung in den Blickpunkt des Bewusstseins. Apperception und Aufmerksamkeit sind dasselbe." J. Baumann, "*Wille und Charakter*," etc., Berlin, 1905, p. 26.

We cannot attend as we *will*, just as we cannot think as we *will*, but *must* think or attend just as those ideas which happen to be present enjoin. Both

as in the matter of ideas already spoken of, and here too the emotional tone (or interest) is an all-important factor. In "attending," the ideational neural charge is conducted into the muscles giving their "setting." And, again, our span of attention is on the whole limited to a harmonizing conceptual system, to associative or correlative ideas. We can however oscillate rapidly between two or more systems and thus in a way attend to more than a single thing;<sup>33</sup> but here the intensity of the one must not be greater than that of the other, for intensity in one direction means fading of attention in the other; so, for example, one cannot attend a toothache with intensity and follow a play at the same time.<sup>34</sup>

In the limelight of our attention, however, hundreds of conscious contents may enter and thousands of sensory stimuli be perceived<sup>35</sup> if they do not disturb the harmony of the associations, or militate against the motor attitude in which we have been "set." Of course those impressions which would lead to contrary motor innervation are here out of the question, their portal and

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attention and thinking are dominated by the momentary associative grouping of ideas and hence our volition is not in reality free, but only appears to be so. See Ziehen, *Psychology*, *loc. cit.*, pp. 212, 235, 202-4. Münsterberg's *Die Willens handlung*, Freiburg, 1888. See also note on page 21.

\* It appears that with practice (which doubtlessly involves the automatic) one can learn to attend to two or even three conceptual systems at the same time, as the experiments of M. Paulhan showed, *Revue Scientifique*, vol. 39, p. 684 (quoted by James). Senator Depew also used to tell of a telegrapher he knew, who, could take and send a message at the same time, or even receive several messages while ticking off another. Whether this was absolutely true or only one of the Senator's remarkable stories, I am not prepared to say. A similar tale is told of Cæsar dictating four letters while writing a fifth! Here undoubtedly the process is one of rapid oscillation. See also Wundt's *Psychology*, p. 235-9, and Cattell's *Experiments*, *Phil. Studien*, vol. iii.

\* Vice Versa, a play may make you forget about the tooth or other ache. So soldiers in battle often do not perceive that they have been wounded. See also Forel's "Hygiene der Nerven und des geistes," Stuttgart, 1905, p. 28-29. "Je intensive ich an etwas denke oder auf etwas achte, desto eingeengt ist das Feld meines Bewusstsein, etc." See however on this point James's "Psychology" (one-vol. edit.), p. 217.

\* For an excellent description of the highly diversified and complicated mass of more or less conscious processes of the psychical life at any moment, as for instance, when at the theatre we see the performance of a play, see F. Paulsen's *Introduction to Philosophy*, Eng. translation by Thilly, pp. 125-126.

path to discharge being shut. "To close the path," again to quote Münsterberg,<sup>36</sup> "means to inhibit the idea which demands such action. . . . We can look at the opera, see every singer and every singer's gown, can listen to every word, can have the whole plot in mind, can hear the thousands of tones which come from the orchestra; and yet combine all that in one act of attention, because it all belongs to the same setting of our reactive apparatus. Whatever the one wants is wanted by the others. But if at the same time our neighbor speaks to us, we do not notice it; his words work as a stimulus which demands an entirely different motor setting as answer. Therefore, the words remain unvivid and unnoticed. . . . Every act of attention becomes, therefore, a complex distribution in the reinforcement and inhibition of mental states."

Here we may pause and succinctly review the facts we have endeavored to define and analyze. Hypnotic sleep supervenes upon the suggestion of such in the waking state, by engaging the attention intensely ("expectant attention," belief in the power, skill to impress, technic of influencing sensation by voice, touch, etc., knowledge of and ability to influence the mind's associative dynamics<sup>37</sup>), and thus inhibiting all else not intimately associated (narrowing the field of consciousness), so producing dissociation (inactivating the aggregate memory mass or Ego). The subject now not being able to associate with his entire mental experience, is highly suggestible and *believes almost reflexly*. Ideation precipitates motor or emotive innervation unless such be inhibited by

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<sup>36</sup> Psychotherapy.

<sup>37</sup> "In the proper application of such inhibition and reinforcement lies the skill of the hypnotist, in the after-effects of such influence upon future association of ideas the nature of post-hypnotic accomplishment" (O. Vogt).

It should not for a moment be lost out of mind that the technic of psychotherapy and of hypnosis especially, must be learned just as that of surgery, for instance, and that it is difficult and necessitates considerable practice and close application. The idea apparently in the minds of many of the students that they could go forth and just hypnotize a patient and "make him do things" is absurd. Even for waking suggestion as practised by the psychotherapist so thorough a knowledge of technic and psychology and much else is necessary, that Loewenfeld, writing in the *Münchener medizinische Wochenschrift* of Jan. 18, 1910, p. 121, says, "dass dieselbe den Anforderungen der Praxis gegenüber ganz und gar unzulänglich ist und zu folgenschweren Missgriffen führen kann."

FIG. 1.



Woman in deep hypnosis.

FIG. 2.



Mild hypnosis.

other ideas. In the waking state such ideation is check-mated if found to be false by the criterion Ego; but in hypnosis the Ego, being dissociated, can no longer inhibit the false idea (or where the Ego is not entirely inhibited, and such is the case in a very large number of subjects, the intensity of attention to the suggested thoughts enervates and makes less vivid all other ideas, thus blocking their path of motor action), the subject has lost the power to conjure up inhibitant or inhibiting ideas (his inhibiting ideas are inhibited<sup>38</sup>), hence reacts upon the suggestion.

It will therefore be evident how under hypnosis one may bring a functionally paralyzed limb to contraction (or where in the paralysis of organic nature a functional quota is contained, which is frequently the case, or when there is still some potential innervation left, lessen the paralysis by producing some contraction) or, allaying a functional contraction, how psychalgias may be made to disappear, or real pain be entirely neutralized by withdrawing attention to other parts.<sup>39</sup>

Let me take this little patient who has been sleeping all this while. Possibly you think he has really fallen asleep. I pick him up. You see he does not mind it. I might pass him around to you and have you look at him like a specimen and again return him to me. He would not know of it, nor would it inconvenience him in the least. Yet this is too valuable a "specimen" should he be dropped or upset! So I'll place him here upon the table. If I whisper to him, he answers. He is therefore not asleep. Now I shall show him this colored square (red) and tell him I shall show him green. You see he answers green. I hold my fingers before his eyes and say he shall see nothing. He opens his eyes—and he sees nothing.<sup>40</sup> I place something into his hand and tell him it is something he has never seen before (it is a

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<sup>38</sup> The credit of pointing out these inhibiting processes in hypnosis belongs to Heidenhain. See also Moll, p. 274, also Münsterberg, who believes the characteristic of the suggested concept to lie in its inhibiting the contrary or opposite of such concept.

<sup>39</sup> See also Ziehen's "Psychotherapie," III, p. 681, in Eulenburg and Samuel's larger work, *loc. cit.*; also Oppenheim, *Lehrbuch*, 4th Edit., p. 1102, for the same in the waking state.

<sup>40</sup> This is a negative hallucination. I cannot go into the interpretation of this phenomenon here. Like rapport and post-hypnotic suggestion, it has not as yet been satisfactorily explained.

common lead-pencil) I ask him to feel it carefully. Now, what is it? He answers, "I do not know." He even looks at it, but doesn't recognize it.

I take this second patient; he sits quite limp in his chair; all tonus seems to have left his body. I take both his hands and paste them to his head. I shall ask the Doctor here to pull the arms away. He cannot do it until I give the word, and now they fall away quite limp again. Or I take this young boy, he is a patient I have not seen before to-day. He came because of a headache (examination proved negative). I tell him his headache will be gone on awaking. But now I also paste his hands to his head and you see I can lift him by the arms from his chair; he is in a state of catalepsy. I take the hands away, I awake him; his headache is gone.

But especially is it possible under hypnosis to bring out an emotional tone, to conjure up a memory that is pleasureable and thus captivate a positive or pleasureable tonal feeling. This is a great help in the treatment of melancholy and depressed states (and the neurasthenic is nearly always depressed) and it is very important in the education or re-education of patients whose "psychology" works wrongly. From the very start one makes such patients "feel better." Many tell me that "it feels as if a load has been lifted." I give them a good prognosis and they straightway begin to fulfil it. That little girl with a smile on her face, although her face seems set, I am treating with dreams of happy play, of butterflies and sunshine. She is here not because she is ill, but because she is sullen, morose, cross, and unmanageable at home. She was constantly getting worse and it was feared that she was mentally deteriorating. The greatest difficulty lay in coaxing her to take treatment. I finally succeeded in putting her asleep and began awaking dreams under hypnosis ("dream therapy") dreams which might create pleasureable interest, and thus kindle a little light in her gloomy soul. I have been treating her for a month, and the nurse tells me that she notices a striking change. The child has lost her dejectedness and smiles. You see her associations have been switched into different channels: she is being "psychically re-educated."

Finally it cannot have escaped your notice, that this very

FIG. 3.



Child in deep hypnosis. Showing absolute relaxation under hypnosis of a highly irritable and nervous child. The boy was brought to the clinic because of truancy and his constant wandering from home (Wandertrieb).





state of absolute inactivity, this condition simulating sleep, may itself be of considerable service to us therapeutically. This induced quiescence calms the wrought up, irritated, "hyper-reflexive" patient, quiets his nerves, and acts as a sedative. But it can also act as a narcotic, and so the confirmed insomniac may receive the rest otherwise denied him. So, too, the sorely afflicted patient with general athetosis, and the choreic may be relieved, for you have seen how their movements cease almost entirely while asleep. (See Figs. 1-5.) For such purposes, therefore, we keep the patient under hypnosis for two or three hours, or even longer. Wetterstrand, who was the first to proceed in this fashion, let his patients sleep for days or even weeks (allowing a few brief waking states daily in order to give food, etc.) and thus instituted an absolute "rest-treatment."

Curiously enough, if left to himself, the hypnotized individual falls into a state in which focal concentration ceases, and a condition of general dissociation or disaggregation supervenes. Now the associations are all not severed but apparently loosened. The attention is, as James has said, "dispersed"; the subject though conscious, thinks really of nothing, and mental activity is at a low ebb. Though some subjects soon awaken, if not suggestively treated, others may be kept in this condition for a long period, or through suggestion be led into normal sleep, or of their own accord fall into such. This condition may alter some, and be very similar to that between wake and sleep, when we easily fall into dreaming and yet know we are dreaming, even seem able to lead or swell our fancies or change them. It is a state in which there easily pop into mind bygone odds and ends, memory-images of erstwhile scenes, long past and since forgotten. It is therefore in these interludes that psycho-analyses are most propitiously made or so-called subconscious affective complexes searched for.

It would appear that our duty had been but poorly performed, if, in bringing this psychological study to a close, we had not attempted some interpretation of the phenomenon of hypnosis along physiological lines and from the vantage ground of the physiological psychologist. Just here however most endeavors have miscarried, and though the theories advanced are numerous and engaging, they have led to few tenable conclusions. It would scarcely be

of much avail to go into these theories at length, and I shall therefore only touch upon the more important ones and epitomize these, together with such aiding facts as I have been able to find in the literature on this and allied subjects, in the following.

In having the patient concentrate his attention—narrowing the field of consciousness—we cause a small portion of his cortex to be overactive, the larger part inactive. This overactive area is more intensely excitable, more intensely reactionable; it is plethoric, the remaining cortex relatively anæmic (which should also be the case in sleep). According to Vogt (and this theory is held to be the most plausible by Forel), the active neural element, the energizing power, working in the nervous system, which he calls “neurokyme,” (*i.e.*, nerve wave) accumulates in non-active areas and is discharged into active areas. Whenever a centre becomes more active for some reason or other, the neurokyme elements from other centres are switched into it. Thus inhibition of the less active areas is produced. This inhibition is also of nutritive nature, metabolism being lessened here and increased in the active centre. Likewise also there is an increased blood flow to the latter, a decreased flow in the former. In the explanation of sleep (be this the ordinary or the hypnotic) he assumes the existence of certain subcortical reflex sleep centres. Such a centre is that for the closing of the orbicularis oculi, which in turn stimulates a vasomotor centre, causing increasing brain-anæmia and so sleep. These centres however may be stimulated through associations, as of the sleeping room, the undressing, etc., or even the remembrance of former sleep. Suggestion will do the same, and hence the rapid sleep upon suggestion.

A similar neural element is assumed by Wundt, who speaks of it as the neurodynamic flow. In his theory there is a like compensation of energy; that accumulating in inhibited areas being conducted into and concentrated in active areas. There is also a secondary vasomotor compensation. As a result of inhibition the capillaries are contracted, while in the active region they are dilated. To this increased blood supply is again due the increased activity of this region. (On account of this concentrated power in the active area, we have in hypnosis the clearer discrimination, the strikingly exact recognition, etc., etc.) Wundt, however, also assumes an in-

FIG. 4.



Deep hypnosis in girl.

FIG. 5.



Girls in deep hypnosis

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hibition of activity in the regions connected with the processes of volition and apperception, postulating an hypothetical apperception centre which he localizes in the frontal lobes of the brain. This psychical faculty has been rejected as an entirely metaphysical assumption. (See Ziehen's *Leitf. der phys. Psychol.*, 7th edit., p. 202-204; also Moll, *loc. cit.*, p. 287.) As for this frontal localization, modern brain physiology does not substantiate it. (See especially Groszlik, "Arch. f. anat. und phys. physiol., Abb.," 1895, and other literature cited by Ziehen, p. 203.)

Not very different from the above compensatory and vaso-motor theory is that of Cappie (so also, fundamentally, the idea of Brown-Séquard, Fechner, etc.). Braid and Carpenter, and in a manner Hack Tuke, also thought the brain to be in an anæmic condition during hypnosis (so also Heidenhain at first), and Mosso believed he had proved it by his experiments. On the other hand there were those who held to a cortical hyperæmia during hypnosis (so Salvioli, Bouchut, Czerny, Pick, Lange). Cappie thought there might be a plethora in the motor region and anæmia elsewhere. Later Brodmann of Berlin had occasion to make observations on a trephined subject and found that no constant changes in the brain circulation took place; he concluded that the change in the cortical blood supply could not alone account for nor be even reckoned as a constant concomitant of either sleep or hypnosis. Nor did the ophthalmoscopic examinations of Förster and Moll show any difference in the retinal arteries (which usually give the same changes as those of the cortex) during hypnosis. Again Heidenhain was able to induce hypnosis in spite of the subject having inhaled amyl nitrite, thus surely having dilated cortical vessels. Then, too, whether there exist hypæmia or hyperæmia, how can we say whether either is cause or affect? Finally, Kronthal (*Der Schlaf des Andern*, Halle 1907) worked out a theory in which sleep had no connection whatsoever with the cortex, but was a passing condition of all living matter in which most reflexes are weakened or disappear. The psyche is the sum of all reflexes, etc. He alludes to the well-known dog of Goltz which was kept alive for one and a half years after having almost the entire brain extirpated, and this dog slept (save for briefer periods of sleep and wakefulness) just like normal dogs.

Parallel with the vaso-compensation in the above theories goes

that of hypo- and hyper-metabolism (see Stadelmann, p. 93). In the waking state the cells are reactionally at their height. While they are receiving stimulation, metabolism is increased. These cells are producing, "using themselves up." This is called *dissimilation* (catabolism). Thereupon follows a period in which the cell recuperates, works less, and "builds up." This is called *assimilation* (anabolism). In reality both processes are constantly going on. In hypnosis dissimilation is at its height in the active area and at its ebb elsewhere in the brain. In the inactive areas assimilation is increased. In this connection may also be mentioned the theory of "activity and inactivity" of living material (in contradistinction to the physiologist's "work and rest") of Ademkewitz. When the brain cells are working actively we have consciousness while when working inactively we get the states seen in visions, dreams, subconsciousness, etc. (see his "Die Grosshirnrinde als Organ der Seele," Wiesbaden, 1902, Chapter 3).

Another theory (Van de Lanoitte, Pupin) rests upon histological grounds, following the discovery of Golgi and Ramon y Cajal, viz., that the end twigs of a neuron are not continuous with, but contiguous to, the next cell (interlacing with its dendrites). A loosening or dissolving of this contiguity would inhibit or break the conductivity of the neural flow. The actual mechanism of this could be accounted for through the amœboid movements of the dendrites (acting as pseudopodia)—which phenomenon would easily explain sleep, hypnosis and other psychic conditions. The amœboid action of the dendrites possibly worked like the positive and negative chemiotaxis of leucocytes. Thus one could also explain the effects of alcohol or coffee (causing the dendrites to spread out and make a strong contact) and other drugs, as morphine, upon the nervous system. (See Rabl-Burckhardt, "Sind die Ganglionzellen amöboid?" *Neurol. Zentral.*, 1890, No. 7; also, Duval, *Revue Scientif.*, 1895; Pupin, "La Neurone," *Thèse de Paris*, 1896, etc., etc.). Ramon y Cajal even holds that on heightened functional activity, etc., new dendrites may thus be sent out and so form new collaterals. (See Ramon y Cajal, *Allg. Betrachtung über d. Morphol. d. Nervenzellen.*, *Arch. f. Anat.*, 1896, Chap. 3-4.) Bechterew (speaking of metabolism of the cell) believes that strong stimulation will quickly tire the cell and bring it and its dendrites to contraction,

thus occasioning the inhibition we see in such cases. Ramon y Cajal holds that such contraction also occurs in the glia-cells, which cells form the contact between ganglion cells, and through contraction, break such conduction. The contraction is brought about by substances extruded from the ganglion cells during their activity (see W. v. Bechterew's "Psyche und Leben," 1908, Chapter xxx).

This highly interesting theory would be extremely convenient in the explanation of many psychic states. It has not been accepted however, because of its being too fanciful to be consistently applied to mental activity in which the simplest process involves not one but many groups of ganglion cells working coördinately together. One could scarcely conceive of the millions of cortical cells in constant motile activity, the dendrites intra- and extra-lacing—as would be expected in the thousandfold play of thoughts—the whole cortex in fact a moving mass (see Hirschlaff, *loc. cit.*, p. 227). Besides, the very foundation upon which the above neuron theory rests has been shaken by the studies of Held, Dogiel, Apáthy, Bethe, and Nissl, who showed that the real elements of the ganglion cells were neurofibrils which traverse both cell and axon and continue on through the next cell, thus forming a continuous neural system (facts already claimed by earlier observers). Bethe even demonstrated (in the crawfish) the possibility of reflex action in the nerve without its ganglion cell, while Nissl described such fibrillary elements as originating outside of the ganglion cell, the so-called "nervous gray" in which both dendrites and axons end, and which may form an intermediary between the axis cylinder of one neuron and the cell of another. Upon this subject (and adverse to the neuron theory) a large literature has sprung up, while on the other hand more recent writers have again begun to question the fibrillary theory (Lenhosesék, Hoche, Schmaus-Saki, etc.). The subject therefore still remains a somewhat mooted one. Finally and in connection with the above, we may mention, without going into detail, Schleich's theory based upon the neuropathia as the inhibiting medium, and Preyer's theory, namely that fixed attention causes an accumulation of waste products which induce the loss of activity in the remaining cortex.

And now after reviewing a considerable body of psychological and psychophysiological literature as well as the data of the neurologists and the books on hypnosis, etc., and after considering



this material and sifting pertinent occurrences in daily life, we have arrived at the end of our study and must ask "Have we interpreted our subject? Has Hypnosis been explained?" Far from it. And yet we have come to a better understanding of the phenomena involved by defining the words of our definition, by dispelling the mysterious and preternatural by instancing analogies in normal mental life, thus dismissing fanciful and mysterious hypotheses and levelling our facts along the highways and byways of psychology. Certain chapters of the subject we have avoided entirely, as their discussion would have carried us very far afield and would have ended in no satisfactory solution (such as rapport, amnesia, negative hallucinations, etc.). We have seen that on the whole the physiological theories have left us in the lurch, while on the other hand where psychological hypotheses have been drawn upon (as in the case of Wundt's apperception centre) they themselves have not been acceptable and have therefore made the subject no simpler. How can we account for the rapidity of effect, the mere phrase causing an inhibition or again revoking it? How can we explain the almost instantaneous production of dissociation? I say "sleep" to the patient with whom I am *en rapport* and his entire psychic condition is altered. I say "awake" and the somnambulistic state fades—into the forgotten! How, asks Hirschlaff, can we interpret the suggested hallucination which is evoked with a word, fleetly changed, or annulled? Neither blood-flow nor neurodynamic waves suffice to explain the enigma, nor any theory based on fatigue products. Far less can we throw light upon the phenomena of negative hallucination and amnesia. This of course does not mean that we should plunge into the metaphysical nor stumble over our shortcomings into the spiritualistic. This as scientists we decry and entirely eschew. The fact is, our science has not as yet advanced far enough to entirely clear up the heart of this as of many another mystery—sleep for instance, or hysteria. And therefore we may bear Wundt's advice in mind, here at the end of our analytical survey, that we need no new forces beyond the physiological and psychological to interpret the phenomena of hypnosis, amazing as they may at first appear, only the laws of the mechanics of the nervous system and the mind are not even yet sufficiently known to suffice for the comprehension of the waking state.

## DIFFERENTIAL DIAGNOSIS BETWEEN EPILEPSY AND HYSTERIA AND THEIR MUTUAL RELATIONSHIP \*

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ORDINARILY there is little difficulty in differentiating hysterical convulsions from epileptic fits. But occasionally the matter is a very difficult one, and I suspect that this difficulty presents itself more frequently than is commonly supposed. Bratz and Falkenberg,<sup>1</sup> two physicians connected with the great Epileptic Colony at Wahlgarten near Berlin, analyzing the records of 2500 patients from that institution, reach the conclusion that a positive diagnosis can always be made if time enough be given; but they admit that in some cases the diagnosis must remain undetermined for a considerable length of time.

The term hystero-epilepsy has been employed to indicate an extremely severe hysterical seizure bearing a marked resemblance to epilepsy; but to many members of the profession the term is wrongly interpreted to mean a seizure which is partly hysterical and partly epileptic in character, one which is a mixture of the two diseases. This conception is nowhere accepted by neurologists; it would be better if the term hystero-epilepsy were discarded altogether. But it is admitted by some that a single patient may have at one time a hysterical attack and at another time an epileptic seizure. This view is held, for example, by Binswanger and Gilles de la Tourette, each the author of a comprehensive monograph on the subject of hysteria.

Somners<sup>2</sup> makes the following classification: (1) Epilepsy which symptomatically resembles the severe forms of hysteria; (2) hysteria which symptomatically resembles genuine epilepsy; (3) epilepsies to which hysteria is added, as a result of the organic cause of the epilepsy; and (4) hysteria in which epilepsy occurs as a pure

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complication. Hysterical manifestations are not uncommon in cases of brain tumor; these same cases may exhibit also epileptic seizures of one sort or another. Thus we see that the same underlying cause may produce both epilepsy and hysteria. Putnam and Waterman<sup>8</sup> remark: "There is no single mark, neither the apparent state of consciousness during the attack, the circumstances of its occurrence, nor the condition of the pupil or knee-jerks, which can be regarded as a pathognomonic sign. Each case must be studied in all its features, and between the seizures as well as during them." The question in differential diagnosis may arise as between a hysterical seizure and a grand convulsion or petit mal of epilepsy; for hysterical seizures may resemble one or the other more or less. No positive rules can be laid down for the differential diagnosis, and we must depend upon our general knowledge of the two diseases. All other things being equal the larger the number of signs of epilepsy present the more likely is the case one of this disease; and the same can be said of hysteria. Of all the signs of epilepsy the one which has always seemed to me the most convincing and which argues most strongly for that diagnosis is the biting of the tongue; yet now I have some reason to question whether it may not happen, if indeed only rarely, that the tongue may be bitten in hysterical seizures. Certain it is that seizures which at first appeared to very competent observers as epileptic have afterwards been considered by the same observers as hysterical. The presence of consciousness is by no means a certain criterion; for occasionally it is present, or only partly lost in epilepsy. And on the other hand, in some hysterical seizures it is partly or wholly lost. It must be remembered that a semiconsciousness or dream state is a well recognized mental state in profound hysteria. An important point mentioned by Putnam and Watermann<sup>8</sup> must be emphasized, viz., that in doubtful cases the behavior of the patient between attacks must be studied and weighed in reaching a conclusion. Moreover, the history of the case is of importance, and the mode of onset, the rate of progress, the frequency of attacks, etc., must all be taken into consideration. One of the most striking features of hysteria is its suggestibility; so it might be concluded in doubtful cases that when the attacks are subject to suggestion they are hysterical in character. And yet I am not satisfied that this is an entirely safe guide, although it ought

to be given much weight. I have reason to believe that occasionally, to some limited extent at least, epileptic seizures are influenced by suggestion. I recall very well the case of a boy I saw at the Mercy Hospital some years ago who was having two or three seizures a day which appeared to be of the ordinary type of essential epilepsy. After the examination by me and talking to, which seemed to impress the boy strongly, judging by his remarks afterwards to the Sisters, he was entirely free from seizures for the next three weeks when he left the hospital. Wetterstrand <sup>4</sup> and Bramwell <sup>5</sup> claim to have much influenced epileptic attacks by the use of hypnotism.

With this brief introduction, I wish to present for your consideration brief records of four clinical cases.

CASE I.—A girl aged 18 was brought to me August 13, 1904, with a history that for one and a half years she had been subject to spells of hiccoughs; and that for one year past she had been subject to convulsive seizures occurring at intervals of a week or two and always at night, during which attacks she stiffened out, frothed at the mouth, and often bit her tongue, and after which she always slept heavily. For three months past she had been subject to hiccoughs without interruption. Whenever she awakens at night she at once begins to hiccough.

Her present illness appears to have begun with sudden loss of voice which occurred in January, 1903, about one year before I saw her. For a period of seven weeks this loss of voice continued; and during this time she was able to whisper but not to speak in her proper ordinary voice. After she had been thus affected seven weeks, the voice in its full natural tone suddenly returned to her one day after her brother had been teasing her; and she has since had no return of this trouble. After this, at irregular intervals, she was troubled with hiccoughs, on account of which she was brought to me.

The spasmodic respiratory actions which I am calling "hiccoughs" for the sake of convenience were really not of the ordinary type of hiccough. The sound was like that of inspiration while the muscles of the larynx were in spasm. These spasmodic respiratory sounds came in pairs usually, but occasionally in threes. (For example, I counted nine pairs or trios of such inspiratory spasms during one minute.) During the time her voice was lost she did not suffer from these respiratory spasms.

Examination discovered irregular loss or diminution of pain sense and marked contraction of the visual fields. Simple experiments in the way of suggestion soon discovered that these spasms could be influenced by these procedures. And after a few visits to my office (I was unable to persuade her to go to a hospital) she was discharged, August 27th, after she had been under observation only 15 days, quite free from respiratory spasms. Five months later her family physician reported that there had been no return of the spasms and that the girl seemed quite well.

Subsequent reports informed me that the girl was in good health and free from spasms and seizures of all sorts until July, 1908, i.e., about four years

after I had discharged her. Later, however, I was informed that on January 16, 1907, she had been subject to convulsive seizures since the preceding July and that these seizures occurred in the daytime as well as at night. She had had one convulsive seizure in July and several since that time, generally three or four weeks apart. Five weeks ago she had two convulsions in the daytime and one attack at eleven o'clock at night. Following these attacks she fell into a deep sleep, upon awakening from which she was stupid and sick at the stomach. These convulsive seizures were of short duration and accompanied by loss of consciousness. After the first fit in July, 1906, there was a pool of blood beside her pillow. All her spells were alike in character.

These spells begin by the eyes turning upwards; then the spine is arched, the muscles are tense, and the patient becomes unconscious, foams at the mouth, usually bites her tongue and is seized with convulsive movements in the arms and legs. At first she is pale and afterwards she becomes red in the face. She sleeps after each attack six or seven hours and awakens feeling very sore. The convulsive seizures last about five minutes, during which time she is entirely unconscious. In one attack she fell while washing the dishes and struck her head, hurting the right eye. Before each attack she feels dizzy. She stated, "I must get hold of somebody; I feel I must; I would be all right if I could get hold of someone."

I put the girl on treatment appropriate for epilepsy and consisting of careful hygienic regulations, bromides, Fowler's solution, etc.

On July 19, 1909, the report reached me that she had had no attack for three months after I last saw her; but had had several of the usual character in the last two weeks. She was again put on treatment.

August 3, 1910 (about one year later), the report was brought to me that she had had no attack since the last note until one day about two weeks ago when she had four attacks, all of the usual character. She bit her tongue and frothed at the mouth in these attacks.

Family History: The patient's father died of consumption. Her mother is rather delicate; and a brother was an epileptic.

What is the correct interpretation of this case? My own view is that it is one of a girl who has exhibited both epileptic and hysterical phenomena. The girl strongly impressed me as being of the hysterical temperament. The sudden loss of voice, the sudden return, the hiccoughs or spasmodic respiratory attacks, both of which phenomena were so strongly influenced by suggestion, and the sensory phenomena with contracted visual fields can leave little doubt that these attacks were hysterical in character. And just because I was so strongly convinced of the hysterical character of these attacks I was at first strongly inclined to the belief that the convulsive seizures, which appeared, you will recall, three years after the onset of the aphonia and eighteen months after the cessation of the hiccough attacks, were also hysterical in character despite the evidence

pointing to essential epilepsy. But with the irregular recurrence of the convulsive seizures and the history that they occurred at night as well as in daytime and were attended by biting of the tongue, frothing at the mouth, pallor and then redness of the face, and that they were followed by deep sleep there is hardly any doubt for the conclusion that these were genuine attacks of essential epilepsy.

CASE II.—An unmarried girl aged 20 was seen by me March 27, 1907. The patient comes of neurotic parents and she has been badly raised. She has always had her own way and never been opposed by her parents. She bosses everybody at home. She has two sisters and one brother and was always considered the healthiest one of the family. The girl is quite pretty except for bad teeth and she has many beaux; but she quarrels with each in turn and sends him away. She worked as telephone operator for one month. She had stopped school three years before. She was always of a nervous temperament but was considered quite well. Her menses began at the age of 15 or 16 and have been regular ever since.

Her present illness dates from August 10, 1905, according to the family. On that day she went to a party; and while sitting in a rocking-chair rocking she fell backwards off the porch to the pavement (several feet) striking her head on the pavement. She was unconscious for a few minutes but got up and stayed at the party and danced afterwards. She could not sleep that night. There was a cut at the occiput and one at the top of the left ear and some discoloration of the left ear. She cannot remember if there was any bleeding at the left ear.

She went to work the following day at the telephone office where she continued regularly at her duty for the next two weeks. Then one morning she went down stairs to collect some bills. At the bottom of the stairs in front of a crowd of men she turned around five or six times. She said "everything seemed to go around." The men caught her and carried her up stairs. She said she was unconscious for one and a half hours and stiff all the time; "they couldn't bend me." The patient remembers turning around but not the subsequent period of this attack. Yet despite this statement she mentioned that five or six doctors were sent for and that she "knew everything."

A couple of days later she returned to work and continued at work for two weeks when the next attack occurred which she describes thus: "I commenced to scream and looked at the door; then I fell to the floor and bumped my head and then I did not remember any more. I think I was unconscious for more than an hour." While she was "unconscious" she knew people were around her. She bit her tongue in this attack until it bled. She was taken home after this attack where she slept. She has not worked since this attack. She stated that two days afterwards instruments were inserted to take her urine from her.

A third and similar attack occurred a day or two later. After this seizure she had seizures every other day for a short time; and then they occurred more and more frequently and were of shorter duration, until the attack lasted only a few seconds. The seizures were always preceded by a scream.

She says that at first she knew all that was going on in her spells, but that she does not lately. Asked if she had bitten her tongue she replied "All the time." She has had as high as twenty attacks in a day. She has spells every night and cries and kicks. Her aunt states that in her "spells" she throws her feet straight up in the air and "in less time than you can get to her it is over with her spell."

I first visited the patient March 27, 1907, at the home of her aunt in the city (patient's home is in Ohio); I was told that she had had seven spells on the previous night and that as soon as she lies down a spell comes on at once or within five minutes. The aunt informed me that if the patient is having a spell and is not being noticed she pinches very hard anyone within her reach. The patient speaks of having many bad dreams during the last year. Often in these dreams she seems to be going down. She said she has seen people flying up above her. And also in spells "I could see a woman who scares me a great deal." For a time last winter she had no fits in the daytime. She skated and went to the theatre and enjoyed much social diversion. Last summer in her attacks she would fall over without screaming or kicking. She has consulted eight doctors.

At the present time a regular gag is used to prevent the patient from biting her tongue. She jumps a great deal in her sleep, which is fitful and disturbed. She thinks of the spells all the time.

The patient has a good appearance and good color. The physical examination is negative. The girl is evidently notional and spoiled. The aunt informs me that if a young man who lives next door, and whom the girl dislikes, passes the window she never fails to have a spell. The girl is noted for her stubbornness and obstinacy. The aunt assured me that she would have a spell within ten minutes after I was gone, and begged me to cure the spells and give the girl a good disposition.

The aunt carries a cushion about the house that the patient may fall easily, and when she does not carry the cushion the patient carries it herself. She gets spells as soon as she lies down. I requested her to lie down and have a spell in my presence that I might witness one. Her reply was "I can't get a spell while you are looking at me." I went to the telephone to make arrangements for her to go to the hospital and while at the telephone the patient took a spell. This spell consisted in convulsive movements lasting only a few seconds, during which she kicked her legs high in the air, at right angles to her body, very much exposing herself. There was no change in her color. In half a minute from the onset of the fit she was about and as lively as ever. This examination was conducted at the home of her aunt; and immediately afterwards I sent the patient to the Allegheny General Hospital for observation.

At the hospital, where she remained for two weeks, she had about two fits a day. One night she bit her tongue; at least there was blood on the pillow and a cut on her tongue. At the hospital, the character of the patient became evident. She was selfwilled and notional, and accustomed to having her own way.

May 9th, Dr. Howell, her family physician, wrote me that she was having spells at the rate of seven or eight a day; and on September 10th of that same year, that she was having three to eight spells a day, light and heavy ones.

This case has puzzled me a great deal. There is much about it that suggests hysteria. The remarkable character of the first two attacks and the fact that the fits appear to be to some extent at least subject to suggestion; the character of these fits themselves, legs kicked high in the air at right angles to the body; and finally the general character of the girl are all suggestive of hysteria. But on the other hand the frequency and character of the attacks, their occurrence at night and finally and chiefly the fact that the tongue is bitten in some of the attacks would seem to argue strongly for epilepsy. Even now my mind is not fully made up as to the character of these attacks; but in a tentative way I hold to the view that they are, all of them, hysterical rather than epileptic, and this despite the history of biting of the tongue on one single reliable observation—that at the Allegheny General Hospital.

CASE III.—A girl aged 11. Since the age of six she has been subject to fits. These fits were always light. But during the last year or so, she has also been subject to heavy convulsions. In a fit at first the child would come running towards her mother with outstretched arms crying "I can't help it!" She would clutch hold of her mother very tight. She would get pale in the face and the lips would become blue, but she was not unconscious. The mother says the attacks lasted only a second and she would immediately afterward run back to play. The attacks come suddenly and without any excitement leading up to them. The attacks, at first, were irregular in occurrence. She might have half a dozen in a day or only one a day. From the first some of the attacks occurred at night and in her sleep. As time went on the attacks became much longer in duration, but she did not seem to be unconscious. She never fell over in a fit. After she had been having these spells for several years she began to sleep after them. She wanted to eat a great deal and she put food into her mouth very quickly. She never bit her tongue; she never fell over in convulsions; and in all of the day attacks she ran to her mother. About six weeks ago she had two severe attacks at five o'clock in the morning, about a week apart, in which the mother thought the girl would die. These attacks lasted approximately 15 minutes each (the girl sleeps with her mother), and she was profoundly unconscious. Immediately afterwards she went to sleep. When she came to she did not know she had had these attacks. She has never passed her water in bed in an attack.

The mother says that the child is a good obedient girl and not hard to control. (The reverse of this seemed to me to be true.) She has not attended school for the last year. She would go to school for two months and stay away for two months. The child is truthful; the mother says that she has no bad tendencies, but that she is fidgety and restless.

Since admission to St. Francis Hospital the patient has had from two to five light seizures a day. The child behaves in these attacks about as the mother had described. In an attack she runs crying, "Mother! Mother!" To the



Sisters and nurses at the hospital she seems to be conscious in most of her attacks. But she has had two or three attacks of apparently much more severe and profound character, in which she was seized with convulsions which lasted for several minutes and she appeared to be profoundly unconscious. In one of these attacks she bit her tongue. The child begged to go home with her mother when visited by her a few days ago.

March 3, 1910. This child has from three to six or eight fits a day. The nurses report that in the attacks she becomes red in the face and is unconscious. The general behavior of the child to my mind suggests hysteria rather than epilepsy. On the other hand it is reported that she has had several attacks in the middle of the night.

The manner of the girl, quick, alert, and mischievous, is that of hysteria rather than epilepsy. I myself witnessed one of the brief attacks, which did not impress me as being one of *petit mal*. There appeared too much fussiness in it; and the child did not look as though she were unconscious in the fit.

August 8, 1910. The little girl left the hospital after a stay of about five weeks. Since the last note, in one convulsion, she bit her tongue. The attacks occur with great frequency, several in a day. Some attacks seem to be influenced by suggestion, and she does not appear to be unconscious in them; while in others she appears to be profoundly unconscious and bites her tongue and the color of the face changes.

This little girl was very fussy, extremely loquacious, and badly spoiled; in short, a very great nuisance to have about. The short attacks from which she suffered when she runs crying, "Mother! Mother!" (in which she does not appear to be unconscious), would impress most observers, I think, as hysterical in character; the general character of the little girl would seem to bear out this view. But it must be remembered that we have a reliable history, that the girl has suffered convulsive seizures during which she was profoundly unconscious and that these attacks have been followed by heavy sleep and that she has bitten her tongue in them. In other words they bear all the marks of true epileptic attacks.

I was at first much inclined to regard this case as one of hysteria; but subsequent observations and reflections lead me to believe it is one of true epilepsy and that the minor attacks despite the somewhat atypical character are those of *petit mal*. The general character of the child might be explained by supposing that she was badly spoiled and badly trained, and a generally forward child.

CASE IV.—School girl, aged 18, first seen March 19, 1910. At birth she was a very large baby; she was a large child. She grew rapidly and was quite tall at 16 years of age. She first saw her menses at the age of 16; and then a whole year elapsed before the second menstrual period; since this time she has seen them every three weeks. She was well as a child and grew rapidly. At

the age of 12 she began to have headaches; these grew more and more severe and more and more frequent during the next three years. At the age of 15 they were extremely severe. When the headaches were most severe she would have suppression of the urine; at least there would be no flow for long periods. Once she went 40 hours without urinating. At the age of 15 she was blistered over the small of the back; this blister produced an ulcer which ate into the back, and for some time ulcerated, and sloughed off, leaving a large hole. Her headaches were at the back of the eyeballs chiefly, although she had some pain at the top of the head. After the age of 15, the headaches were for a time better and then worse; and on the whole she was somewhat relieved. But since last October (five months) the headaches have never ceased and she has been during this time out of school on account of them. They are located at the back of the eyeballs.

Her first menstrual period was in April and the following August when she was 16 years of age she suffered a convulsive seizure which her mother describes as follows:

The patient came running from the kitchen calling out, "Rub me! Rub me!" and kept calling out for five minutes, when she fell over frothing at the mouth, choking. She was red in the face and afterwards very white. She was entirely unconscious during the seizures, which lasted two hours.

The next fit occurred the following Christmas and the next in the following August. Both were like the first one. Since last October she has had a fit regularly two or three days before each menstrual period. The headaches are on the increase and are worse at the time of the fits.

The patient always has a warning of some sort before a fit. Lately the warning has been in the shape of voices which she hears particularly in the right ear. One warning she speaks of is the ringing of bells, like a telephone bell, and a tingling which accompanies it and seems to be a part of it. It seems as though she could hear the bell down the right side of the body. Once this sensation came on the left side. Before the occurrence of the first fit, on two occasions she came running to her mother calling out "music" as though she heard music in her ears.

She is absolutely unconscious in every fit and in several of them she has bitten her tongue. A heavy sleep commonly follows a fit. She vomits as she comes to, but has not vomited at other times. Her eyesight does not trouble her; and an oculist who examined her recently said that she did not need glasses. She complains much of black spots before her eyes.

The family history is negative. The father is a sound healthy man and has a dozen healthy brothers and sisters. The mother is well.

Examination: The girl is tall, weight 61 Kg. (135 lbs.), height 172 cm. (5 ft. 8 inches); color is good and I can note no scars on the tongue. She seems bright enough and does not possess the mental state suggestive of epilepsy, but rather that which would be in keeping with hysteria. Physical examination at my office is negative. Eye-grounds seem quite normal; pupils react to light and accommodation; vision seems good. Visual fields normal. No areas of anaesthesia; but she is everywhere hyperæsthetic. Heart and lungs appear normal. There is irregular sighing respiration.

She was admitted to the Allegheny General Hospital February 23rd and remained there under observation and treatment until about May 11th. She

complained of black spots in front of her eyes of all kinds of shapes, clouds, tadpoles, etc. She felt a beating in the arms and legs.

March 30, 1910. This morning she heard a bell ringing and had a numb feeling on the right side. This always means a convulsion either that same day or a day or two later. Patient worried about this but since convulsion has not occurred she is more encouraged.

April 2, 1910. Patient complains of ringing of bells in both ears,—three times to-day,—lasting only a few minutes each time. Had numbness for a few minutes down left side (was on right side last time). Patient is nervous and apprehensive. Previously she had shown exuberance of spirits.

April 3, 1910. Patient had five convulsions to-day. The first convulsion came on about 2.30 A.M. and lasted about three minutes. Patient says she could feel convulsion coming on. It was very severe, involving the entire body. It began with a loud outcry; then the patient became very rigid and frothed at the mouth, biting and gnashing the teeth; pupils were dilated; face was very cyanotic. After the convulsion the patient immediately went to sleep and slept about ten minutes, and upon awakening was much dazed. Twenty minutes later she vomited. Patient had five similar convulsions at intervals of about two hours.

April 5, 1910. Patient complains of "back and limbs," says "this is all;" seems cheerful.

April 8, 1910. Head nurse says that patient has not complained of headache in last three days.

April 15, 1910. Complains of pain in the right side in a very insistent way. Examination by Dr. Simpson: She has an infantile uterus and a cyst in one ovary.

April 18, 1910. Pain in side. Probably not as bad as she says it is. Seems comfortable and cheerful, very little headache. No pain in eyeballs. Eating and sleeping well. Nurse's report is good.

April 28, 1910. Better last two days. Complains only of spots in front of left eye, none in right. No definite headache, but a dull ache behind the eyeballs. Was out of bed about four hours.

May 3, 1910. Complains of headache only, says it is very severe. Some catarrhal discharge from head.

May 5, 1910. Warnings like the sound of a telephone bell about twelve o'clock last night; had them about an hour. Feeling fairly well. Has headache and is depressed.

May 7, 1910. No "warning" or other "aura"; feeling better. Slight headache yesterday. Not menstruating yet.

May 10, 1910. No convulsion. Patient cheerful; feels convulsion will not come. Some headache. Has not menstruated.

May 11, 1910. Has gone as long as two weeks without headache. One week ago had "warning" but no fit. Discharged.

During her stay at the hospital the nurses were carefully instructed never to inquire as to the headaches. While she did complain of them more or less, whole days would pass and once a period of two weeks in which she made no complaint of headache. And

this leads me to believe that the statement of her suffering constantly from headache for five months before I saw her was very much exaggerated. The manner of the girl in the hospital was in the main demonstrative and emotional. For example, she kissed and embraced the head nurse and each of two under nurses on her birthday; and as she grew fond of one or the other she would desire to kiss her. She was either exuberant in spirits or depressed. Her depression impressed me as not having any very great depth to it; as being the pout of a spoiled girl.

The general temperament and manner of the girl strongly impressed me as being hysterical. But on the other hand the convulsions which were observed at the Allegheny General Hospital bear most of the marks of true epilepsy, and yet they lack a little something. For example, the sleep of only ten minutes after a severe convulsion is unusual and one would expect much longer and heavier sleep. The auræ (if they really were such) are also peculiar in several respects. The description of the first fit as given by the mother is suggestive of both hysteria and epilepsy. The aura which occurred when fits did not follow is another peculiarity of the case.

Again it is to be remembered in this case that severe headaches are a prominent feature, that they came at the age of 12 and increased until the age of 16, when she suffered her first convulsive seizure. The study of the case makes it reasonable to suppose that both headaches and convulsive seizures are an expression of one underlying cause. It seems reasonable to assume that the case is not an organic one in the ordinary sense of the word. Despite the resemblance of the fits to those of true epilepsy, I am much disposed to regard them as hysterical in character.

Two letters received from the patient, the first on August 19, 1910, and the second one on September 19, 1910 (after this paper had been written), sufficiently explain themselves and scarcely need comment. It does not occur to me that the views I have expressed of the case need any revision in the light of these letters; but they do raise the question as to the propriety of removing the cystic ovary.

"I have had only one spasm since I came home and that was on July 8th. I had warnings before it came on. It was not nearly as hard as the others I have had. I have had several warnings without the attacks. This month

I did not have any warnings before my menstruation, but this week I have had warnings every day since Monday [four days]. There has just been one week since I came home that I have not had the headaches and I have been home since May 28th. The pain is all on the top of my head and back of my eyeballs. The pain never ceases. My head never stops aching any more. Those black spots are always in front of my eyes. This week my side has been hurting me so bad. When I got up this morning I could not put my limb down, it is so sore to touch and such sharp pain goes through it all the time. Sometimes it is worse than others. I have gained ten pounds in flesh but have no strength in my limbs. When I go up or down stairs everything gets black in front of me. When I first came home I thought I was cured. I am clear discouraged now. I am troubled a great deal with leucorrhœa."

The last letter received reads: "It has been some time since I have written to you, and I guess it is because I am feeling so much better that I forgot to write. My head and side have not been hurting me for two weeks. That has been the longest since I came home from the hospital. Those black spots are still in front of me yet, but I am much stronger. I weigh 145 pounds."

To summarize briefly my conclusions regarding these four cases:

The first case is one presenting both hysterical manifestations and true epileptic fits.

The diagnosis in the second case is still in doubt; but I lean to the view that the seizures from which the patient suffered are hysterical in character.

The third case, which at first impressed me as being hysterical in character, appears to me as one of true epilepsy.

The fourth case is probably one of hysteria; and there is possibility here of underlying organic disease which may stand in etiological relationship to the fits.

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- <sup>3</sup>Putnam and Waterman: Boston Medical and Surgical Journal, 1905, clii, 509.
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# Pathology

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## THE RELATION OF THROMBOPHLEBITIS OF THE PORTAL AND SPLENIC VEINS TO SPLENIC ANÆMIA AND BANTI'S DISEASE

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THE acceptance by American physicians of splenic anæmia as a definite clinical entity was largely the result of the interest excited by two papers published by Osler in 1900 and 1902, in the *American Journal of the Medical Sciences*. In the latter paper, particularly, Osler, upon the ground of the fifteen cases observed by himself and an analysis of forty-five cases of anæmia with enlarged spleen seen by other American writers, argued in favor of splenic anæmia as a distinct chronic affection, probably an intoxication of unknown origin, characterized by a progressive enlargement of the spleen, anæmia of a secondary type, leucopænia, a marked tendency to hemorrhage, particularly from the stomach, and in many cases a terminal stage with cirrhosis of the liver, jaundice, and ascites (Banti's disease). Lyon, in his article on splenic anæmia in volume four of Osler's "Modern Medicine," accepts this definition as covering the main features of the disease, and this conception of the affection is generally accepted in this country.

Osler regarded the conditions described separately in the literature under the various heads of primitive splenomegaly, splenic anæmia, splenomegalic cirrhosis of the liver, and Banti's disease as representing respectively the early and late stages of one and the same malady; and this view has been accepted by practically all the later American writers, including Lyon, whose chapter is one of the most recent upon the subject (1908). In Osler's analysis of the cases observed by himself and others there is, however, no mention made of one of the most important pathological features of

Banti's disease, insisted upon by Banti himself, namely, the changes in the veins of the portal system. Likewise, Lyon in his chapter in "Modern Medicine" does not emphasize the occurrence of changes in the portal and mesenteric veins as constituting any important feature of the Banti pathological complex. He summarizes the pathology briefly as consisting of a hyperplasia and fibrosis of the spleen, anæmia of a secondary type, and a terminal cirrhosis. In a short paragraph he refers to changes in the portal veins as originally described by Banti, and more recently by Dock and Warthin in this country, but treats them as incidental features and does not assign to them any place of special importance in the pathology of splenic anæmia, although stating that the complete picture of splenic anæmia may be caused by obstruction of the portal and splenic veins, either within or outside of the vessel.

Dock and Warthin (*American Journal of the Medical Sciences*, 1904) were the first American authors to report cases of splenic anæmia showing marked changes in the portal vein and its branches of the nature of a chronic thrombophlebitis with stenosis and calcification. These writers studied two cases representing early and late stages of Banti's disease. Both cases showed a similar fibroid hyperplasia of the spleen, associated in one case with an early hepatic cirrhosis, in the second with an advanced stage of this condition. A very thorough study of the pathology of these two cases is given by Warthin, who regarded the extensive changes in the portal vein and its branches as a most important pathological feature and raised the question concerning a possible relationship between these vascular conditions and the splenic fibrosis. He believed that the changes in the spleen of the two cases showed no features that could not be explained by a chronic passive congestion of marked degree; that is, such a fibroid hyperplasia may follow a severe and long-continued passive congestion, as is sometimes the case in the lung or liver.

Both cases reported by Dock and Warthin presented the following common features: splenic fibrosis, hepatic cirrhosis, stenosis and calcification of portal vein, chronic passive congestion of portal system, sclerosis of portal radicles with marked tendency to hyaline change and calcification throughout the portal area, hyperplasia of hæmolymp-nodes and bone-marrow and new formation of

lymphoid tissue in the retroperitoneal fat, and secondary anæmia. In one case the splenic fibrosis and the hepatic cirrhosis were not so far advanced as in the other case; but in both the splenic condition seemed older than the hepatic. The question then arises, is the splenic fibrosis primary or is it secondary to the obstruction of the portal and splenic circulation following an old thrombophlebitis of the portal tract? As to the cause of the latter condition, this may have been a primary involvement of the veins, or it may have been secondary to some toxic condition of the portal blood dependent upon changes in the spleen or intestinal tract, or a general toxæmia. The authors find themselves unable to decide, and leave the question open with the conclusion that there is a possibility that the clinical picture of splenic anæmia or Banti's disease complex may be caused by a stenosis of the portal or splenic veins following an old thrombophlebitis.

Unfortunately the condition of the portal vessels had not been noted in the American cases analyzed by Osler, and he does not mention it in any one of his fifteen cases. Since the study of Dock and Warthin, few cases of splenic anæmia or Banti's disease have been reported in this country. I have been able to find but one case in which the condition of the splenic vein was described. Sanford and Dolley (*American Journal of the Medical Sciences*, 1905) give a clinical and pathological study of a case which they regard as an early stage of splenic anæmia. The anatomical findings were dislocation of the spleen with marked elongation and torsion of its vessels, and thrombosis, chronic passive congestion, and fibrosis of the spleen, atrophic cirrhosis of the liver, chronic passive congestion of entire portal tract, compensatory hyperplasia of hæmolymp-nodes, hyperplasia of bone-marrow and secondary anæmia.

Sanford and Dolley believe that the enormous elongation and torsion of the vessels possessed a probable etiological significance in this case. The recent thrombus found in the splenic vein was the cause of a sudden enlargement of the spleen occurring shortly before death. The writers regard this case as one of splenic anæmia, associated with a cirrhosis of the liver, inasmuch as the fibrosis of the two organs appeared to be equal in degree. The portal vein is described as normal, but the intima of the splenic vein as somewhat



irregularly thickened, being composed of dense fibrous tissue with few nuclei. The adventitia showed a more pronounced thickening, and there was some fibroblastic proliferation about the vasa vasorum. The splenic vein and its branches contained soft mixed thrombi, in places loosely attached to the intima. Organization of the thrombi was found to be well advanced in places. The veins in the pancreas were also noted as having thickened walls. No calcification was observed.

From the description given by these writers there can be little doubt that their case was one of Banti's complex, showing the sclerotic changes of the portal tract as emphasized by him as constituting one of the essential features of the complex.

In Germany, Lossen in 1904 (*Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, Bd. 13) gave a detailed study of a case of splenic anæmia, without cirrhosis, but showing widespread sclerosis and thrombosis of the portal system. The veins were dilated, varicose, in part thrombosed, showing a thickened and hyaline intima, with patches of calcification in some vessels. The thrombi were of different ages. The spleen showed the characteristic fibrosis, but no increase of the reticulum was seen in the liver. Lossen calls attention to the fact that the pathological complex of splenic tumor, liver cirrhosis, and phleboscclerosis of the portal system, emphasized by Banti as the essential triad of the disease which bears his name, has not received sufficient attention in the literature. Most of the German writers have doubted Banti's interpretation of his cases; on one hand these are regarded as cases of primary hepatic cirrhosis with an unusually large splenic tumor; on the other hand, the changes in liver and spleen are referred to a common cause. The acceptance of the Banti complex as a distinct entity has not been as general in Germany as in America. Lossen attempts to settle this question by an analysis of several cases of splenic anæmia observed in the Königsberg clinic and a large number collected from the literature. From this analysis he is not able to draw any certain conclusion as to the unity of the relations of the three different factors of the complex and the anæmia and the leucopænia. He concludes that there are co-ordinated affections of the spleen, liver, and veins of the portal system that owe their origin to some common unknown

cause. Hypertrophy of the spleen, no matter of what origin, appears to cause in the majority of cases a diminution in the red blood-cells and hæmoglobin, associated in many instances with a leucopænia.

The discussions concerning the entity of splenic anæmia and Banti's disease have been almost wholly concerned with the clinical symptoms of splenic enlargement, secondary anæmia, hæmatemesis, etc. The pathological studies so far published of cases showing the clinical picture of splenic anæmia or Banti's disease show a remarkable diversity in the pathological findings, so that many authors doubt the individuality of these conditions and express the conviction that different affections have been classed under this term. In this country we find Dock and Warthin expressing the view that the symptom-complex of splenic anæmia represents a group of varying pathological conditions, the splenic enlargement being secondary. Stengel, also (*American Journal of the Medical Sciences*, 1904), reports "Varieties of Splenic Anæmia." As seen above, Lossen inclines to the belief that the symptom-complex of secondary anæmia, leucopænia and splenic enlargement may be the result of different causes; but that the hepatic cirrhosis, the fibrosis of the spleen, and the endophlebitic changes in the portal tract are associated or co-ordinated affections due to a common cause.

Banti's insistence upon the occurrence of an old phlebitis in the portal system as one of the cardinal features of the disease called after him, and the cases of this kind reported by Dock and Warthin and by Lossen make it highly important that the etiology and significance of thrombophlebitis of the splenic veins should be determined in the hope of settling the question of the individual entity of the Banti triad. If the symptom-complex and pathological complex of splenic anæmia and Banti's disease can be the result of different factors causing thrombophlebitis of the splenic or portal vein, the nosological position of the two so-called diseases will be settled.

Throughout the literature there occur observations of cases showing enlargement of the spleen associated with thrombosis of the portal or splenic veins. The earliest case is that of Morgagni, "Epis. xxxvi," an observation of Valsalva. A young man about

twenty years of age, originally strong and healthy, became during two years very pale, and complained of slight disorders in belly and thorax. A large tumor was discovered in the left hypochondrium, with a sense of weight and a difficulty of respiration on walking. To these symptoms there was added suddenly a large vomiting of blood, with a great loss of strength, an increase in the size of the tumor, and fever. Under treatment the vomiting stopped for three months, but the tumor remained constant in size, and the skin pale. Two or three attacks of hæmatemesis then occurred, high fever developed and death took place. The autopsy showed the presence of a very marked anæmia, a greatly enlarged spleen weighing four pounds and a half, and larger than the liver. Deep within its convex surface could be felt two hard, firm bodies of the size of a large nut, which were regarded as bony concretions. The liver was pale, mottled with black spots. In the trunks of the splenic vein polypoid concretions were present, following the branches of the vein, even into the substance of the spleen.

In the absence of our modern blood examination this description of Morgagni still reads much like a case of splenic anæmia. That cases of marked splenic enlargement, with dilatation of the vasa brevia, leading to hemorrhage from the stomach, were not unknown to the older medical writers is clearly shown in Morgagni's discussion of this case and his references to the literature.

Frerichs, in his "Clinical Treatise on the Diseases of the Liver," 1858, records observations on a number of cases of sclerosis or thrombosis of the portal vein associated with enlargement of the spleen, one of which, "Observation xxix," suggests a possible splenic anæmia. In the chapter on thrombosis and pylephlebitis he gives an analysis of the reported cases of portal thrombosis, referring the most frequent cause of obstruction of the portal vein to cirrhosis of the liver and chronic atrophy of this organ. He states that the character of the thrombus in such cases is usually that of an obturating thrombus extending from the trunk into the radicles. The vein wall usually shows traces of chronic inflammation and frequently contains calcareous plates. He quotes the following cases:

Foisson (*Gaz. des Hôpitaux*, 1848).—Man aged fifty, symptoms of intermittent fever, pallor, icterus, frequent vomiting, and ascites. Spleen very large; liver small and hard. Walls of portal veins completely ossified;

lumen filled with a firm reddish mass, extending a long way in the form of a semi-solid coagulum.

MANNERET (*Union Médicale*, 1849).—Man, aged 42, suffering from hæmatemesis, bloody diarrhoea and ascites. Liver atrophied, not granular. Spleen very large. Portal vein enlarged, walls rough and thickened, lumen filled with a firm reddish and, in some places, whitish thrombus.

DOWEL (*Dublin Quarterly Journal of Medical Science*, 1851).—Reports two cases of thrombosis of portal vein and radicles, in one case associated with thickening and calcification of the vein walls. Granular atrophy of the liver was present.

GINTRAC (*Observations et Recherches sur l'Obliteration de la Veine Porte*, 1856).—Reports five similar cases. In two cases of atrophic cirrhosis the portal veins were completely blocked with thrombi, while the spleens were of normal size. In a third case of cirrhosis the wall of the portal vein contained calcareous plates, while the vessel at its bifurcation was partly blocked by a brown, firm coagulum.

Frerichs also quotes an observation made by Craigie, who, in one instance, found the portal vein constricted as far as the splenic and mesenteric veins.

Botkin (*Virchow's Arch.*, Bd. 30, 1864) gives a detailed report of a case showing the complex of anæmia, bloody stools, hæmatemesis and enlarged spleen. Autopsy showed an enlarged spleen, with thickened capsule and firm consistency. The liver was small, its surface somewhat furrowed and granular. The portal vein and its branches were completely filled with an old colorless thrombus, thickest at the bifurcation, and extending as far as the hilum of the spleen, but becoming smaller in the mesenteric veins. In the liver a marked atrophy was present with an increase of the periportal connective tissue. Botkin argues the relationship between the portal thrombosis and the hepatic cirrhosis, and advances the idea that the cirrhosis may follow the thrombosis, instead of the reverse process, as is usually held to be the case. In the absence of knowledge as to the blood condition, this case fulfils the other requirements of the Banti complex.

Still more striking in its identity with the modern conception of splenic anæmia is the case reported by Andrew Clark for Dr. Moorhead (*Trans. of London Pathological Society*, xviii, 1867). A man, aged 57, of sallow complexion, had repeated attacks of bloody stools and hæmatemesis, causing death. The spleen was found to be three times its normal size, while the liver is described as being thinner than usual and anæmic, but otherwise presenting

a normal appearance. The wall of the portal vein was found to be calcified and the lumen occluded by a thrombus. At the site of the thrombus the vein was dilated, while on the hepatic side of the obstruction it was contracted, resembling a fibrous cord. This thrombus was regarded as the immediate cause of the hemorrhages from the stomach. Clark considered this case to be unique because of its extreme rarity, and states that no similar case is mentioned in the works of any modern author, and that there is no mention of any thrombosis or embolism of the portal vein causing hæmatemesis.

Spiegelberg (*Virch. Arch.*, 142, 1895) reports a case of thrombosis of portal and part of splenic vein, bloody diarrhœa, recurring ascites and œdema, great enlargement of the spleen, liver not cirrhotic, walls of portal, splenic and mesenteric veins sclerotic and in part calcified. Spiegelberg reviews the literature, abstracting the cases of Gintrac, Foisson, and Andrew Clark given above and gives the following new cases:

**RAIKEM** (*Mem. de l'Acad. Royale de Méd. de Belgique*, 1848).—Soldier, 57 years old, suffered a year and a half with œdema, ascites, diuresis and diarrhœa. Portal vein obliterated and walls contained numerous hard, bone-like plates.

**VIRCHOW** (*Würzburger Verh.*, vii, 1857).—Man, aged 66 years, suffered from icterus, ascites and œdema. Spleen greatly enlarged, liver small, Varicose anastomosis between splenic and azygos veins. Portal vein obliterated by a calcified thrombus the thickness of a finger. Splenic vein dilated toward the spleen, covered with calcareous plates toward the portal end.

**BALFOUR AND STEWART** (*Edinb. Med. Jour.*, 1869).—Man, 20 years of age, sudden gastric pain and hæmatemesis, and later œdema and ascites. Splenic tumor with thickened capsule. Liver atrophic, aneurism of splenic vein. Portal vein obturated by thrombus. Walls of both portal and splenic veins atheromatous and calcified.

**DURAND-FARDEL** (*Bull. de la Soc. Anatom. de Paris*, lviii, 1883).—Woman aged 45. Cachexia and ascites. Splenic tumor. Portal vein and radicles thrombosed to the hilum of liver. Splenic vein sclerotic, dilated and filled with clots. Calcareous plates in wall of portal vein.

Spiegelberg was unable to decide as to whether the thrombosis in these cases was primary or secondary to the calcification of the vein walls. It accompanied the latter process only in about half of the cases. In his own case he regards it as a possibility that the thrombosis was the primary process causing the changes in the vessel walls. The enlargement of the spleen he regards as the

result of the stasis in the portal vein; and considers the occurrence of hæmatemesis, œdema, ascites, and diarrhœa as easily explicable. As to the cause of the thrombosis he has no knowledge, and he regards the disease and its anatomical findings as a rare observation.

Borrmann (*Deut. Arch. f. klin. Medicin*, Bd. 59, 1897) made a detailed study of all the reported cases that he could find in the literature up to date, including those of Gintrac, Botkin, Balfour and Stewart, Raikem, and Andrew Clark (reported under the name of Morehad, the attending surgeon (Moorhead) for whom Clark reported the case before the London Pathological Society). He adds abstracts of the following cases not yet mentioned in this paper:

OPPOLZER (*Oester. Zeitschrift f. prakt. Heilkunde*, vii, 1861).—Patient 37 years old, intermittent fever for thirteen years, then a period of improvement, followed by six months of repeated attacks of hæmatemesis, ascites. Death from anæmia and exhaustion. Ulcer and carcinoma of stomach were excluded and a diagnosis of portal vein thrombosis was made before death. Autopsy showed an enlarged spleen and small liver. Portal and splenic veins dilated and atheromatous, containing many thrombi almost wholly obliterating the lumen. Thrombi in part old and dry. Hemorrhagic erosions in mucosa of stomach and intestine.

PIPPOW ("Ueber die Obturation der Pfortader," *Inaug. Dis.*, Berlin, 1886).—Case presenting dyspeptic symptoms for half a year, then rapidly progressive ascites and splenic tumor. Autopsy showed the portal vein to be obliterated and converted into a connective-tissue cord. The obliteration was due to the organization of a thrombus still demonstrable in the radicles of the portal vein. No cause for the thrombus discoverable.

OSLER (*Jour. of Anat.*, 1882).—Case of obliteration of the portal vein in a man, aged 28 years, apparently healthy, who felt weakness and a dull pain in upper portion of the abdomen. Frequent hæmatemesis, pulsation in epigastrium, spleen enlarged, liver small. On autopsy spleen greatly enlarged, adherent, parenchyma firm, connective tissue thickened, some calcification. Arteries of spleen tortuous, walls thickened and calcified, in part also thrombosed, and at the hilum showing small saccular aneurisms. Portal vein obliterated and converted into a fibrous cord 2 cm. (0.8 inch) above its origin; its main branches also closed. All the radicles of the portal vein showed localized thickening and calcification, and contained thrombi. Liver small, capsule thickened, no cirrhotic changes. Other abdominal veins greatly dilated.

LEYDEN (*Berl. klin. Wochensch.*, 1866, Nr. 13).—Patient 57 years old, repeated hæmatemesis, splenic tumor, ascites. Diagnosis of thrombosis of the portal vein without liver cirrhosis, because of sudden vomiting of blood and rapid return of ascites without icterus or any decrease in the size of the liver. Autopsy showed large spleen, great enlargement of stomach veins, liver small and hard but showing no increase of connective tissue. Portal

vein filled with a colorless thrombus mass, which, toward the liver, was fibrous and calcified at its periphery. Wall of portal vein markedly thickened, intima in places firmly attached to thrombus. Tissues surrounding the portal vein hard, indurated, tendon-like.

ALEXANDER (*Berl. klin. Wochens.*, 1866, Nr. 4).—Patient 36 years old, had pneumonia nine years previously and three years after had sudden discharge of blood from mouth and anus, and had three attacks of this in the next six years at intervals of two years. Weak and anæmic, pains in upper portion of abdomen, dyspeptic symptoms, death with symptoms of peritonitis. Autopsy showed splenic tumor and an old thrombosis of the portal vein extending into all of its radicles, particularly into the mesenteric veins, in some of the branches the thrombi reaching the intestine. Splenic vein for some distance from the portal also thrombosed. No trace of cirrhosis in the liver. Writer emphasized the importance of the diagnostic significance of the intermittent bleeding from mouth and anus.

To these cases Borrmann added two new ones, one from the clinic of Eichhorst and the other from the private practice of Seitz, of Zürich:

CASE I.—Woman, aged 63 years, weak, œdema, ascites, splenic enlargement, death from collapse. Autopsy showed the presence of syphilitic hepatitis, gumma, thrombosis of the portal vein, spleen three times normal size and adherent. Thrombosis of portal vein regarded as the result of syphilitic disease of the vein-wall.

CASE II.—Man, aged 63 years, pale, complaining of loss of appetite, thirst, vomiting, general weakness, attacks of abdominal pain, diarrhœa; finally vomiting of blood, death from collapse. Autopsy showed obturation of portal vein by an old thrombus; wall of vein thickened, these changes also seen in some of the portal radicles and these also contained fresher thrombi. Liver showed typical syphilitic changes. The thrombosis of the portal vein was regarded as the result of a primary syphilitic disease of the wall of the vein.

In all Borrmann collected eighteen cases of portal thrombosis from the literature which with his two cases he grouped according to etiology as follows: (1) syphilis (4 cases: Jastrowitz, Bülan, Loewenfold, Borrmann); (2) chronic peritonitis with connective-tissue strands about the porta (3 cases: Achard, Frerichs); (3) gallstones (1 case: Key); (4) swollen glands in the porta with syphilis of liver (1 case: Coco); (5) apparently unknown etiology (11 cases: Gintrac, Oppolzer, Balfour-Stewart, Raikem, Moorhead, Osler, Leyden, and Borrmann's second case).

In the first nine cases the cause of the thrombosis was probably the compression of the portal vein from without, although in several of these cases undoubtedly several factors aided. Of the

eleven cases of apparently unknown etiology eight showed marked changes in the wall of the portal vein (sclerosis, atheroma, calcification, etc.) comparable to those found in arteriosclerosis and atheroma of the arteries. In three of the cases the condition of the vein-wall was not reported.

Borrmann then discusses in full the occurrence of sclerotic changes in the portal vein, showing the relatively few references to it in the literature. The occurrence of marantic thrombosis in the portal vein is also discussed (Nonne: *Deutsch. Arch. f. klin. Med.*, 1885). The question of the occurrence of a general phlebosclerosis also is taken up. From the whole discussion Borrmann concludes that:

1. There occurs a primary sclerotic and atheromatous disease of the walls of the portal vein, and more rarely of its radicles (splenic, mesenteric) of unknown etiology.

2. As a result of this disease of the vein-walls thrombosis of the portal vein may be produced.

3. There exists an independent portal thrombosis, having no connection with a liver cirrhosis or other conditions in the neighborhood of the portal vein, but due to primary disease of the wall of the portal vein and circulatory disturbances of unknown etiology.

In regard to the etiology of primary portal sclerosis and atheroma, Borrmann holds that the condition is wholly analogous to the atheroma and sclerosis of the aorta, and that the same etiological factors of the latter process hold good in the case of the portal vein, syphilis in particular.

Insofar as the diagnostic features of the condition are concerned the cases analyzed by Borrmann show that portal thrombosis can occur in relatively healthy men with sudden pain and symptoms of extreme passive congestion in the portal system (ascites, hæmatemesis, splenic tumor, bloody stools, etc.). The liver may remain normal or undergo a moderate atrophy, with or without increase of the connective tissue. The sudden and severe onset of the symptoms and the rapid return of the ascites after puncture are especially significant of obturation of the portal vein. Naturally the course of the symptoms will depend upon the degree of obliteration of the portal lumen. If canalization of the thrombus occurs, or if the lumen of the vein is partly or wholly opened up, the course may



be long. In some of the cases the thrombosis is undoubtedly progressive and successive openings and closings of the vein may occur.

Saxer (*Centbl. f. allg. Pathologie u. path. Anatomie*, Bd. 13, 1902) reports a case of portal thrombosis in a male, 22 years old; symptoms of hæmatemesis, weakness and anæmia. Death from gangrene of intestine due to thrombosis of vessels. At autopsy the entire portal system, main trunk, splenic and mesenteric veins, were found thrombosed, the thrombi in the smaller radicles more recent, in the main trunks older. No ascites, marked splenic tumor, liver small, no cirrhosis, gangrene of intestine. No apparent cause for thrombosis. Condition of vein-walls not described.

Saxer considers critically the effects of portal thrombosis, and from the published cases concludes that some degree of atrophy of the liver is the most constant finding. No specific changes in the liver characterize the condition. He does not accept Borrmann's conclusions as to the primary nature of the disease of the vein wall, and does not regard portal phlebosclerosis as a probable cause of portal thrombosis. To establish such a connection between the two processes he believes that the condition of phlebosclerosis must be demonstrated in cases in which severe clinical and anatomical changes have not occurred as the result of the portal stasis. Lossen (see above) has fulfilled this condition in the case reported by him, so that Saxer's objection does not hold.

Saxer also reports a case of thrombosis of the superior mesenteric artery and vein with beginning gangrene of the intestine. In this case the venous thrombosis is undoubtedly very recent and secondary to the changes produced by the embolism, and the case need not be considered in this connection.

From the cases reported in the literature it would seem that portal thrombosis is not extremely rare, that it was known to the older medical observers, and that its symptomatology is sufficiently well-defined to make possible a diagnosis during life. Summing up the results of our analysis of the reported cases we find the older ones reported under the head of portal thrombosis, enlarged spleen and hæmatemesis, while the more recent cases are included under the designation splenic anæmia or Banti's disease. With the exception of the blood-condition (secondary anæmia and leucopænia)

there is absolutely nothing in the clinical or pathological complex by which the cases of portal or splenic thrombosis can be separated from the cases of splenic anæmia or Banti's disease. Moreover, the blood condition is only a recent diagnostic factor, and some of the recent cases show that secondary anæmia and leucopænia do occur in the cases of thrombosis. In both conditions we may have precisely the same complexes: splenic tumor, secondary anæmia, leucopænia, hæmatemesis, chronic course, cirrhosis or atrophy of the liver, ascites or none, etc.; indeed, every factor of the symptom-complex that Osler lays down as the criterion for splenic anæmia is also a factor in the complex of portal sclerosis and thrombosis. We are certainly justified therefore in doubting the accuracy of Osler's conception of splenic anæmia as a distinct individual affection. If the symptom-complex of splenic anæmia can be produced by thrombosis, compression, twisting etc., of the portal or splenic veins, as the reported cases show; and if cases without portal or splenic vein obstruction show the picture of splenic anæmia also, then there must be wholly different affections grouped under the term, and a differentiation is necessary.

The questions to be settled are: Can simple obstruction of the portal or splenic veins produce the symptom-complex of splenic anæmia or Banti's disease? This question is apparently already answered by some of the cases reported. Is the thrombo-phlebitis of the portal tract the primary condition, or is it caused by a common factor producing the splenic tumor, liver changes, etc.? What is the cause of the secondary anæmia and the leucopænia found so nearly constantly in these conditions? Are these conditions the result of a definite intoxication or simply the results of the secondary disturbance in the spleen?

These questions are so important that they demand investigation in order that the nosological position of splenic anæmia can be determined. Since the paper of Dock and Warthin (1903) there have occurred in the service of the pathological laboratory of the Medical Department of the University of Michigan three cases showing the characteristic complex of splenic anæmia, in two of which cases complete obliteration of the splenic vein was present. In the third case, one in which splenectomy was performed, the condition of portal and splenic veins could not be fully determined

at operation, but the portion of the vein at the hilum showed a recent thrombophlebitis. These cases in connection with two relatively recent cases of thrombosis of portal and splenic veins of known etiology are hereby reported in the hope of throwing some light upon the questions raised above. A series of animal experimental investigations has also been carried out for the same purpose and the results are included in this study. The new cases are as follows:

CASE I.—M. S., aged 47, widower, woodworker, American, was admitted to the University Hospital (Dr. Dock), Jan. 12, 1904, complaining of weakness and shortness of breath.

His father died at 61 of apoplexy; his mother at 84 of old age. Two brothers and one sister are living and well; seven other brothers and sisters died in infancy and childhood, one each of whooping-cough, "water on the brain," and typhoid fever, the other causes being unknown. He does not know of any nervous, constitutional, blood, or malignant disease in the family. Is the father of seven children, all in good health.

Patient has had mumps and whooping-cough in childhood. He had "fever and ague for four years," from 8 to 12 years of age. He had typhoid fever at 27 years of age, lasting 3 months, but it left no effects so far as he knows, except that he never weighed as much as before. His former weight was 150-160; after typhoid 135-140.

Habits of eating show nothing noteworthy; denies alcoholic abuse and venereal disease.

The present disease began four years ago with dizziness and nausea. There was a feeling of coldness in the stomach, followed by the bringing up of small quantities of blood. There was no actual vomiting. These symptoms lasted two or three weeks, during which the stools were black. The stools were regular and of normal consistence. There was a second similar attack soon after the first, and then none until two years later, another a year later, and another two months ago. All resembled the first attack and all were followed by shortness of breath. Two months ago, after the patient had been working at his trade, dizziness came on at night and he spit blood. About the same time he noticed a swelling in the abdomen. Two nights later he had a severe hemorrhage from the stomach, amounting, he thinks to three pints. The swelling increased, reaching a larger size than it is now. Three days later he vomited some bitter fluid mixed with blood. The stools were dark after these hemorrhages and sometimes looked like clotted blood. Jan. 1 there was an attack like the first one described above.

The appetite has always been good, bowels regular.

*Status præsens* (Dr. R. S. Morris): Temperature, 98.3; pulse, 96, respiration, 24.

Patient is 5 feet 9 inches in height; of medium frame. Weighs 130 pounds (stripped). The muscles are small and soft, panniculus thin. The reflexes are normal, joints normal; lymph-glands not enlarged.

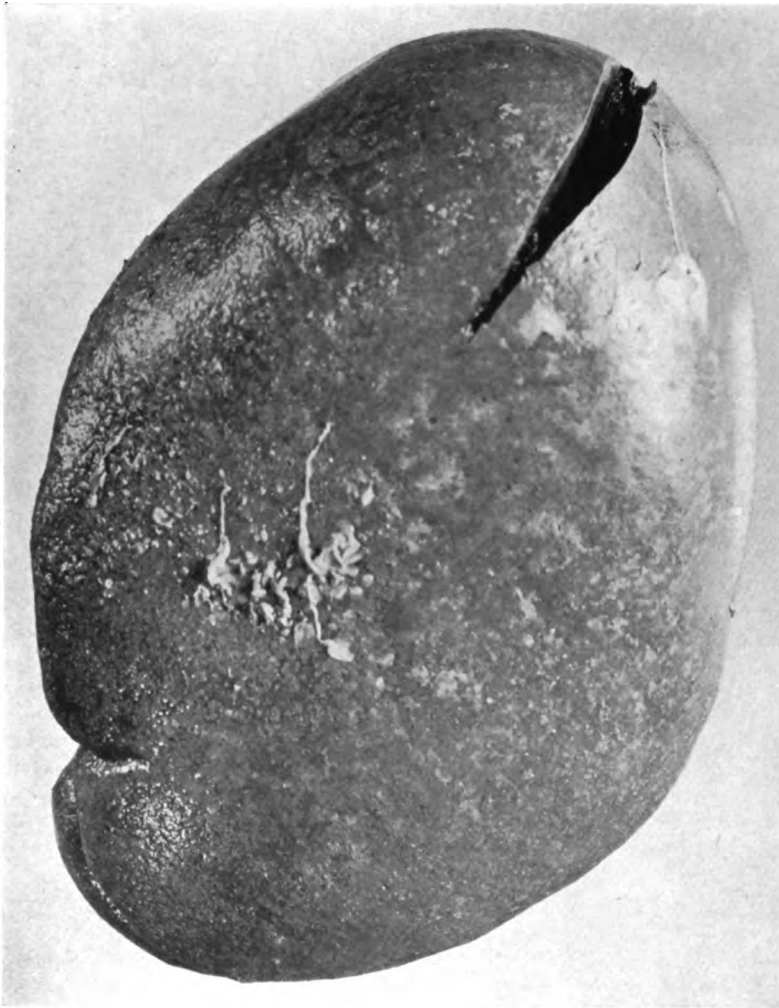
Patient is able to be about, his expression is suggestive of weakness and anxiety; the face is more emaciated than the body. The skin is sallow

**FIG. 1.**



**Spleen from Case I, splenic anæmia with thrombosis of portal and splenic veins.**

**FIG. 2.**



**Spleen from Case II, splenic anæmia with thrombosis of portal and splenic veins.**

all over, but not yellow; dry and smooth, fairly elastic. There are small pigmented spots on the shoulders, back, abdomen and forearms, right groin and external genitals. No œdema. The scleræ are clear, conjunctivæ and lips pale, no trace of icterus on hard palate or beneath tongue.

The teeth are in extremely poor condition; in the lower jaw only the incisors are left; the upper incisors are gone; the other teeth are worn and caseous; the gums are swollen; and have grayish detritus about teeth; mucosa of the mouth pale; tongue pale, clean, moist.

The thorax is broad, deep, clavicles prominent; the lower aperture enlarged. Expansion is slight; the thorax moves as a whole. Percussion of the lungs shows higher pitch in left anterior. There is harsh vesicular breathing in that part. Vesicular breathing weak in the lower part, otherwise negative. Lung-liver line on the fifth rib, nipple-line, moves freely in respiration.

Apex beat in the fourth interspace, 1 cm. (0.4 in.) outside nipple-line. Dulness begins at upper edge of fourth rib, extends 1 cm. (0.4 inch) beyond nipple-line and to left edge of sternum. First sound at apex weak and impure, second sound clear. At the second left interspace there is a short soft blowing murmur following the first sound; the second pulmonary sound is accentuated; in the tricuspid area the first sound is flapping and impure. Pulse of moderate size; tension and rhythm good, rate high.

The abdomen is prominent  $2\frac{1}{2}$  cm. (1 inch) above the level of the ribs; measures 35 inches; is distended in the flanks, especially the left, and above the umbilicus. The swelling above the umbilicus, filling up the epigastrium, shows a transmitted pulsation; it feels tense; there is no peristaltic motion in it. No succussion. The umbilicus is not prominent. The abdominal veins are not enlarged. The walls are tense and resistant. There is no fluctuation. On "dipping," a mass can be felt below the left costal border, extending to the parasternal line and to the level of the umbilicus. The surface of the mass is smooth. An edge can be felt only in the upper part, also a notch. Palpation otherwise negative. The abdomen gives a tympanitic note on percussion over the top and right flank; in the left side, below the anterior axillary line, there is diminished resonance, quickly clearing up when patient lies on the right side; sitting up, dulness appears in the lower part of the abdomen, to within two finger-breadths of the navel. The liver cannot be felt; dulness to the margin of the ribs. The splenic dulness begins in the seventh interspace in the anterior axillary line and continues to the end of the mass described above.

Urine examination. No albumin, sugar or albumose. Sediment, a few red blood-corpuscles; a few hyalin casts with amorphous granules in them.

Blood: flow is scanty; drop, pale, watery. Red corpuscles 1,920,000; leucocytes, 2900; hæmoglobin, 40 per cent. Red blood-corpuscles vary considerably in size; many macrocytes, microcytes, and poikilocytes; many corpuscles take the stain poorly. Differential count: small lymphocytes 5 per cent.; large, 6.5 per cent.; transitional, 1.5 per cent.; polynuclear, 86 per cent.; eosinophile, 0.9 per cent. Examination of stool negative.

Various enemata and the rectal tube had no effect in reducing the size of the abdomen, which remained tympanitic.

Feb. 9th. The distention is less, the abdomen being on the level of the ribs, the epigastric fulness not so marked; the flanks are resonant; the

splenic region is distended; the spleen reaches to the left parasternal line and within 1 cm. (0.4 inch) of the level of the umbilicus. The edge of the spleen is thick, hard; splenic dulness begins in the eighth interspace. The liver dulness extends from the sixth to the seventh rib.

Feb. 10. Test breakfast shows good motor power; slight excess of mucus. No free HCl; combined HCl, 10. No digestion in tube.

Feb. 11. Test meal shows same conditions as to motor power, mucus and digestion as yesterday. No free HCl; combined, 8.

Feb. 23. No material change except in the condition of the blood, which has improved in number of red cells and hæmoglobin, but with low leucocytes and relatively high polynuclear cells. The counts are as follows:

Date	Red cells	Leucocytes	Hæmoglobin
Jan. 13.....	1,920,000	2900	40 per cent.
Jan. 19.....	2,320,000	2546	40 per cent.
Jan. 23.....	2,560,000	2700	40 per cent.
Jan. 27.....	2,840,000	2037	45 per cent.
Feb. 4.....	2,880,000	2900	55 per cent.
Feb. 6.....	3,240,000	4329	60 per cent.
Feb. 10.....	2,920,000	2546	65 per cent.
Feb. 20.....	4,480,000	2300	75 per cent.

The treatment consisted in Bland's pill in ascending doses to 1.6 Gm. (25 grains) *t.i.d.*; sodium cacodylatem, 0.015 Gm. (gr.  $\frac{1}{4}$ ) *t.i.d.*, and dilute hydrochloric acid, 15 drops after meals.

Feb. 23. The patient went home. The diagnosis of splenic anæmia, with variations in the blood condition due to recent hemorrhage, was made at a clinic, Jan. 18. Ulcer of the stomach, cirrhosis of the liver, malignant disease of the stomach, pernicious anæmia, chronic nephritis, and aneurism were especially considered in the differential diagnosis. Banti's disease in the sense that cirrhosis of the liver was also present was considered. The facies suggested cirrhosis, but as the ascites disappeared so quickly and as there was no evidence of collateral circulation the cirrhosis was considered not very far advanced, if at all present. The possibility of endothelioma of the spleen, and of obliteration of the portal vein were also considered, but splenic anæmia was thought to be the least objectionable term to apply. The old history of malaria was not thought to have any near relation to the present disease.

June 19, 1904, the patient returned to the hospital. He said he had been fairly well until May 30. On that day, just before dinner, he vomited a large quantity of blood. About the same time he passed a considerable quantity of blackish blood per rectum. During the next three days he passed blood, and then no more until June 18th, when blood again appeared in the stool.

He now looks pale, the face is emaciated, lips slightly cyanotic. Tongue small, pale, with a thin white coat. The abdomen is about one inch above the level of the ribs, measures 36 inches, the epigastrium and flanks are distended out of proportion, no movable dulness. The superficial veins are not enlarged. The spleen extends 3 finger-breadths below the margin of the

ribs. The heart sounds are strong, accompanied by a harsh systolic murmur, loudest over the base. The radial pulse is small, quick, tension low.

Blood, pale and watery. Red corpuscles, 1,572,000; leucocytes, 3540; hæmoglobin, 18 per cent. (Miescher). The red corpuscles are pale, irregular in size and shape, but macrocytes and microcytes are not numerous as before. Differential count; small lymphocytes, 22.3 per cent.; large lymphocytes 1.3 per cent., polynuclear, 71.8 per cent.; eosinophile (polynuclear), 4.6 per cent.; normoblasts, 3 per cent.

Treatment was at once begun for the hemorrhages. Morphine was given to control restlessness, only a few doses being necessary. Gelatin was given by the mouth and rectum, calcium chloride in 3 Gm. (40 grain) doses *t.i.d.*, with liquid diet after a few days. After two day's treatment there were no more hemorrhages for 37 hours.

June 20. At 10 P.M. the patient vomited about 300 c.c. (10 fluidounces) of blood; 10 or 15 minutes previously he had complained of nausea.

June 21. At 1 A.M. vomited 250 c.c. (8 fluidounces) of bloody fluid, and after taking coffee vomited about 200 c.c. (6 fluidounces). (The vomitus contains meat and a currant eaten yesterday at noon.—G. D.) Since then has taken a glass of ice water without ill effects. He is pale, the hands and feet warm. He complains of pain above the eyes, and the eyebrows are drawn together. The first heart sound is impure at the apex. The systolic murmur is loudest in the second and third interspaces to the left of the sternum. The abdomen shows no change. The lateral superficial veins of the thorax and the right external mammary vein are slightly enlarged. A weak and distant impulse can be felt over the spleen.

June 28. The pulse is at times weak and unequal. Abdomen measures 36 inches.

July 9. Patient takes broth without discomfort. Abdomen measures 35¾ inches. Tympanitic in the flanks.

July 13. Soft diet to replace part of the liquid.

July 19. Patient is pale but says he feels well. Pulse, 96, regular, size medium; blood-pressure (Riva-Rocci), 120. Blaud's pill ordered in increasing doses.

July 20. Patient was allowed to be up in wheel-chair, but soon complained of swelling of legs and abdomen.

July 22. The œdema of the legs has disappeared, but the abdomen is larger, measuring 37¾ inches. The umbilicus protrudes slightly; the superficial veins in middle of the abdomen are enlarged. Resonance in the flanks while lying down, but on sitting up dulness appears above pubes, with fluctuation.

Splenic area exposed to X-rays for 10 minutes.

July 26. Patient still complains of fulness, pain and pressure in the abdomen. Dr. Morris tapped abdomen and withdrew 6550 c.c. (7 quarts) of clear, yellow fluid, sp. g. 1011. The operation gave the patient great relief. Says all the symptoms are relieved. Spleen after tapping easily felt 2½ inches below the costal margin. The epigastric fulness persists, and shows peristaltic movement.

July 27. The patient had been feeling well all day, but was suddenly taken with nausea and vomiting, and threw up altogether about 2 litres (2



quarts) of bloody fluid. He was weak and dizzy afterwards, but slept well, and next morning complained of roaring and buzzing in the ears. The abdomen is filling again.

July 28. Paracentesis, withdrawing 6560 c.c. (7 quarts). Fluid is similar to that withdrawn before. The cells are chiefly small lymphocytes, with few other forms.

July 31. After feeling better, and taking soft diet, the patient began to vomit about 7 P.M. In several vomiting spells brought up about 1 litre (1 quart) of fluid blood and clots.

Aug. 1. Six-thirty P.M., vomited 1 litre (1 quart) of bloody material.

Aug. 3. Patient is becoming apathetic. At times does not recognize relatives. Does not hear well. Refuses nourishment by mouth but retains enemata. Pulse small, weak, frequent.

Aug. 5. Patient seemed stronger in the morning; was conscious and talked about his condition. Took nourishment by mouth. Pulse stronger and regular. About 2.30 P.M. began to get restless, tried to get out of bed, complained of pain in the epigastrium. Vomited a small quantity of bloody mucus. Then grew rapidly weaker, breathing became labored, involuntary micturition. At 4.20 P.M. died quietly.

There was no fever during the later stage.

The urine was free from albumin, albumose and sugar, but at times contained hyaline casts and cylindroids.

The blood counts were as follows:

Date	Red corpuscles	Leucocytes	Hæmoglobin
June 19.....	1,572,000	3540	18 per cent. (Miescher).
July 11.....	1,800,000	2801	25 per cent. (Miescher).
July 18.....	1,760,000	3565	25 per cent. (Miescher).
July 27.....	1,503,000	7117	25 per cent. (Miescher).

The examination and differential counts of the blood in the final period were made by Dr. Ward Ellis and Mr. Smith, student-assistant in the medical clinic. I have gone over the slides and find the same kind of changes as those given in the examination of June 10th. There are many pale cells, with and without vacuoles, a few microcytes, many macrocytes and poikilocytes, a small but increasing number of nucleated red cells, with megaloblasts in the later examinations,—at most 6 per thousand megaloblasts as compared with leucocytes. The leucocytes always showed relatively low lymphocytes, increased polynuclears, up to 83 per cent., few eosinophiles, and degenerates. Mast-cells appeared in about 1 per cent. in the last week.

AUTOPSY-PROTOCOL No. V., 1904, PATHOLOGICAL LABORATORY.—Age, 49; nationality, American; status, widower. Occupation, wagon maker. Day and hour of death, Aug. 5, 1904, 4.20 P.M.; time of autopsy, Aug. 6, 1904, 11.55 P.M.; clinical diagnosis, Banti's disease; ascites; œsophageal hemorrhage; tympanites. Prosector, R. S. Morris.

*External Examination.*—Build: Height, 5 ft. 8 in. Signs of trauma,—paracentesis; wound in left side 6 mm. ( $\frac{1}{4}$  inch) above middle of Poupart's ligament; injection wound over right femoral and one inch below McBurney's point; mucous membranes show extreme pallor. Skin, pale yellow. Muscles,

small; on section, fair color; costal cartilages cut easily; no calcification. Rigor mortis, present throughout. Panniculus, small amount; on section light yellow in color. Oedema, moderate amount in feet and legs.

Thorax: Diaphragm,—right fourth rib; left, fourth, I.C.S. Pleural cavities contain about 400 c.c. (12 fluidounces) of clear yellow fluid smelling strongly of formalin. Pericardium contains about 100 c.c. (4 fluidounces) of reddish yellow fluid with marked formalin odor. Heart, sub-pericardial fat practically absent; no soldier spots in epicardium; slightly larger than cadaver's right fist. On section the muscle is very pale; looks like cooked meat; no clots; no fluid blood. Right heart, ventricle wall,  $1\frac{1}{4}$  cm. ( $\frac{1}{2}$  inch); trabeculae not flattened. Valves, mitral orifice admits two fingers; slight thickening of edge of flaps; aortic orifice admits thumb with ease; tricuspid admits three fingers; pulmonary admits two fingers; valves negative. Left lung, numerous rather firm adhesions over upper lobe and lower anterior and lower border of lower lobes; left lung same as right. Right lung, pleura entirely free; crepitant throughout; anthracosis marked. Section, abundant frothy fluid; no airless areas found. Great vessels of thorax, aorta negative throughout.

Mouth and neck, cervical portion of oesophagus: Just above the cardia there are some enlarged veins, the largest being about 4 mm. ( $\frac{1}{8}$  inch) in diameter. Considerable reddish fluid in oesophagus; no clots. One inch from cardia and dilated vein is a round, punched-out opening the size of a BB shot. Two similar but smaller openings are seen within 18 mm. ( $\frac{3}{4}$  inch) of this the size of a pin-point and pin-head respectively, both being situated over veins. Pressure caused a small amount of blood to escape from opening. The varices extend upward about 4 to 5 cm. ( $1\frac{1}{2}$  to 2 inches) from the cardia. They are most numerous in the posterior wall of the oesophagus.

Abdomen, abdominal cavity: Contains about 1000 c.c. (1 quart) of dark yellowish fluid with odor of formalin. No adhesions except about spleen. Spleen, much enlarged ( $19 \times 13 \times 7$  cm., weight 980 Gm.) and extends forward two finger-breadths from costal margin in nipple-line. Upward seventh I.C.S. in the middle axillary line. Over the lower posterior edge there are fairly numerous, rather firm adhesions. The surface is pale and is covered with many roundish or oval, semi-translucent, slightly elevated bodies, the diameter varying from 0.5 to 2 or 3 mm. ( $\frac{1}{16}$  to  $\frac{1}{8}$  inch). Spleen cuts with increased resistance. The bodies seen on the capsule do not extend into the substance of the spleen. Cut surface yields little blood on pressure, is extremely pale. Malpighian corpuscles are not made out with unaided eye. The main branch of the splenic vein is occluded by a firm, rather grayish-brown mass. Adrenals, negative. Left kidney, fatty capsule poor in fat; fibrous capsule strips easily; surface smooth; cut surface shows marked pallor; cortex and pyramids show same pallor; resistance in cutting seems increased; surface has a cooked appearance. Right kidney,—right is similar to left in all respects. Ureters and bladder, negative. Duodenum and bile passages, negative. Stomach contains about 200 c.c. (6 fluidounces) of reddish fluid; about 500 c.c. (1 pint) of clotted blood; mucosa very pale; shows little post-mortem change; no ulceration; veins enlarged; gastrosplenic veins varicose. Small intestine, slightly distended; impossible for lack of time to open the bowel and examine mucosa. Large intestine, slightly distended; mucosa not

examined. Liver looks small, especially in transverse diameter; left lobe seems particularly reduced in size; the organ is very pale, grayish. On the convex surface of the right lobe there are a few enlarged veins, not quite so large as those of the œsophagus. The lower edge of the liver shows an omega-shaped dip in which the gall-bladder rests. The surface is generally smooth; consistence firm. Cut surface was not examined, the specimen being preserved intact for gross preparation. Gall-bladder, negative. Portal vein, small, hard, wall calcareous. Lumen completely filled with a semi-translucent hyaline or cartilaginous substance. The calcification extends about 9 cm. ( $3\frac{1}{2}$  inches) from the liver. The splenic and mesenteric veins filled with old organized and calcified thrombi extending into some of the smaller radicles. Walls of the veins of entire portal tract greatly thickened and contain calcareous plates. Pancreas small, very firm; veins thrombosed. Hæmolymp-h-glands, prevertebral; very pale grayish pink; size varies from common pea to small lima bean.

*Microscopical Examination.*—Heart, atrophy. Lungs, emphysema; chronic passive congestion; œdema. Spleen, fibroid hyperplasia; atrophy of lymphoid tissue; few follicles; hyaline masses in sites of follicles; stroma greatly increased, hyaline. Veins in part thrombosed, with very thick, hyaline walls. Splenic vein, old thrombophlebitis, with organization and calcification; lumen completely obliterated by organized thrombus; evidences of marked inflammation in and about vein walls; calcification of wall. Portal vein, old phlebitis; all the radicles of the portal system show signs of old phlebitis with sclerosis and calcification; completely organized thrombi in some of the smaller vessels. Pancreas, chronic interstitial pancreatitis; sclerosis of vein-walls; larger veins obturated by organizing thrombi; atrophy of parenchyma. Liver, atrophy; relative increase of connective-tissue; no cirrhosis. Other organs, atrophy, chronic passive congestion; sclerosis of vessels.

*Pathological Diagnosis.*—Thrombosis of splenic vein and branches (splenic anæmia); old thrombophlebitis of portal radicles; fibroid hyperplasia of spleen; atrophy and passive congestion of all other organs; œsophageal varices with erosions; hemorrhage.

**CASE II.**—(Private Case.) Male, about 45 years of age, gave a clinical history of anæmia, leucopœnia, weakness, abdominal tumor, hæmatemesis, ascites, lasting for several years, and finally death from hemorrhage. Further history not obtainable as notes have been lost. Clinical diagnosis of cirrhosis of the liver, probably Banti's disease. Complete autopsy-record not obtainable. Abdomen contained free fluid. Liver small, hard, and nodular. Spleen much enlarged, reaching to median line and to level of umbilicus. Capsule thickened, showing hyaline patches. The splenic vein was completely obliterated and converted into a solid calcareous cord about as thick as a man's thumb. The portal vein was narrowed, lumen obturated, filled with a solid fibrous mass and wall partly calcified. On microscopical examination a fibroid hyperplasia of the spleen was found, and in the liver a well-marked cirrhosis. The pathological diagnosis was Banti's disease in a late stage. No further notes concerning the general pathology of this case are now available. Spleen alone preserved, and its general features will be described fully below.

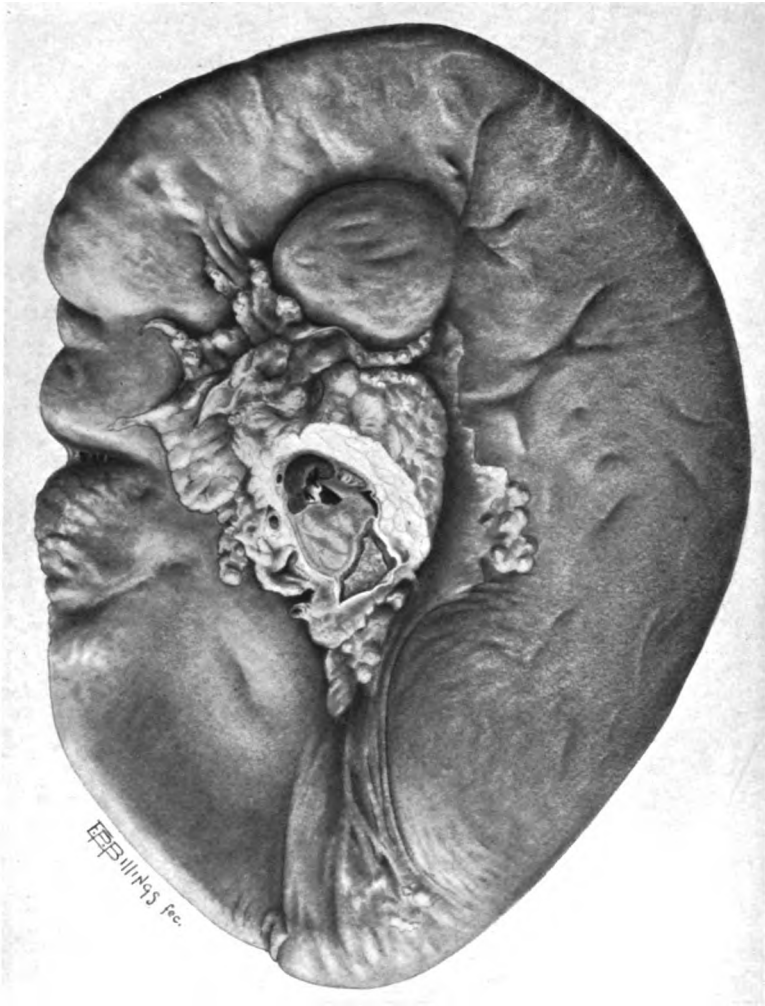
**CASE III.**—Female, aged about 35 years, gave history of increasing weak-

**FIG. 3.**



**Spleen from Case III, splenic anæmia with thrombosis of portal and splenic veins.**

**FIG. 4.**



**Spleen from case of thrombosis of splenic vein, showing obturation of splenic vein by organized thrombus.**

ness and anæmia of several years' standing with occasional attacks of hæmatemesis. No previous history of infectious disease. No evidence of syphilis. Recently abdomen has increased in size, and splenic tumor can be felt, reaching below the umbilicus and to the right of median line. Clinical diagnosis of splenic anæmia. Red blood-cells 3,500,000, hæmoglobin, 60 per cent., white cells 6 to 10,000. After severe hæmatemesis spleen became about one-third smaller. Splenectomy performed. No cirrhosis noted in liver. Condition of splenic vein noted at operation as friable and "diseased." Patient living, improved. Spleen was much enlarged, about three times normal size, but not so large as in preceding cases (16 x 12 x 6 cm., weight, 750 Gm.). Capsule thickened, showing hyaline patches and stringy adhesions. Examination of the hilum showed the remains of an obliterated splenic vein. Anomalous artery and vein to lower pole. Microscopical examination showed a fibrous hyperplasia of the spleen and an old thrombophlebitis of the splenic vein. Character of splenic hyperplasia similar to that in preceding cases, but not so old a process. Pathological diagnosis was Banti's disease, early stage, splenic phlebitis with old thrombus.

**SUMMARY OF CASES I, II, AND III.**—The three cases presented clinically the picture of splenic anæmia or Banti's disease, and were so diagnosed. Case I is a typical case of splenic anæmia; so is Case III, while Case II is an example of Banti's complex. Pathologically, the common feature was the fibroid hyperplasia of the spleen in connection with an old thrombophlebitis of the splenic and portal veins with obturation of the former vessel. The character of the changes in the spleen and in the splenic vein is identical in all three cases, differing only in degree in Cases II and III. Only in Case I was a complete examination of the entire portal tract possible. As in the two cases reported by Dock and Warthin the entire portal system showed marked hyaline thickening of the vein-walls, even to complete obliteration of the lumen in the smallest branches. In the hyaline walls of these completely obliterated veins a deposit of lime-salts was common. Cross-sections of the mesentery often showed numerous calcareous concretions corresponding to calcified veins. Mesenteric lymph-nodes atrophic and contained small calcareous concretions.

In Case I the splenic vein was examined throughout. Its lumen was dilated and filled with the remains of an old thrombus showing a greater or less degree of organization. Microscopical examination showed the vessel-lumen in some portions to be completely replaced by a fibrous cord. Masses and plates of lime-salts were common both in the thrombus and in the vein-wall. The latter was greatly

thickened, but very irregularly so. The tissues outside the vein-wall presented a picture of a very old chronic inflammation with evidence of old hemorrhage in the form of hæmosiderin. The organized thrombus extended into the pancreatic veins, there showing changes identical with those found in the mesenteric veins. The pancreas also showed a condition of chronic interstitial inflammation.

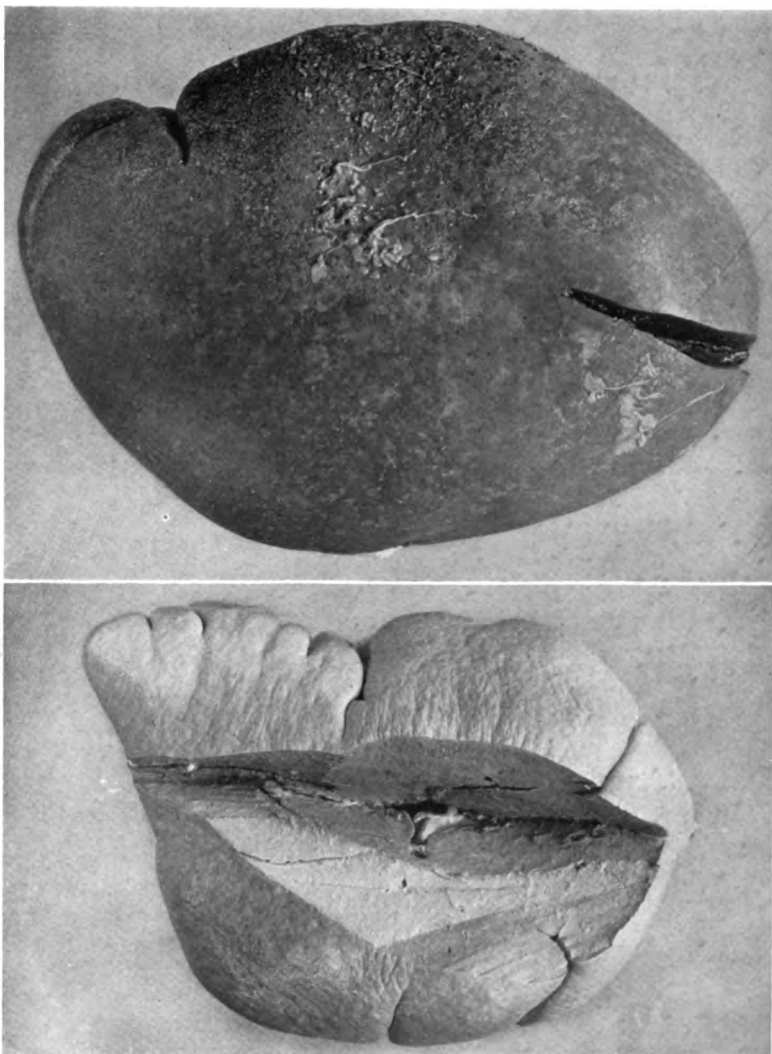
The organization of the thrombus in the splenic vein seemed most complete and older toward the spleen, while the portal portion of the vein showed remains of the thrombus still unorganized and evidently of a more recent date of formation. In this case again as in the two cases described by Dock and Warthin, the thrombosis of the splenic vein and the fibroid hyperplasia of the spleen seemed older than the changes found in the liver. The degree of fibrosis and of the hyaline change point to an older process than the liver condition.

A similar condition undoubtedly existed in Case II, although not absolutely determined. The complete obliteration and calcification of the splenic vein points to an old process, perhaps older than in Case I, since the hepatic condition was farther advanced, taking on the character of a cirrhosis. Case III represents an earlier stage. The obstruction to the portal flow from the spleen was evidently less marked and the inflammatory process in the splenic vein still active, as shown by the slight leucocytosis present, and the splenic hyperplasia less advanced. The smooth liver shows that the obstruction to the outflow of the splenic blood is not caused by a cirrhosis, but that there must exist some vascular condition to account for it.

That the thrombophlebitis is the primary thing is made probable from the sudden development of the symptoms, the small liver and the large spleen. As to the etiology, syphilis seems to be excluded in all three cases. A cryptogenic infection or intoxication in the portal tract may be assumed. It seems justifiable, however, to put these three cases by the side of the two described by Dock and Warthin, as examples of a splenic anæmia complex dependent upon a thrombophlebitis of the splenic vein of unknown origin.

**CASES OF ACUTE INFECTIVE THROMBOPHLEBITIS OF THE SPLENIC VEIN.**—This laboratory has also seen two cases of recent infective thrombophlebitis of the splenic and mesenteric veins asso-

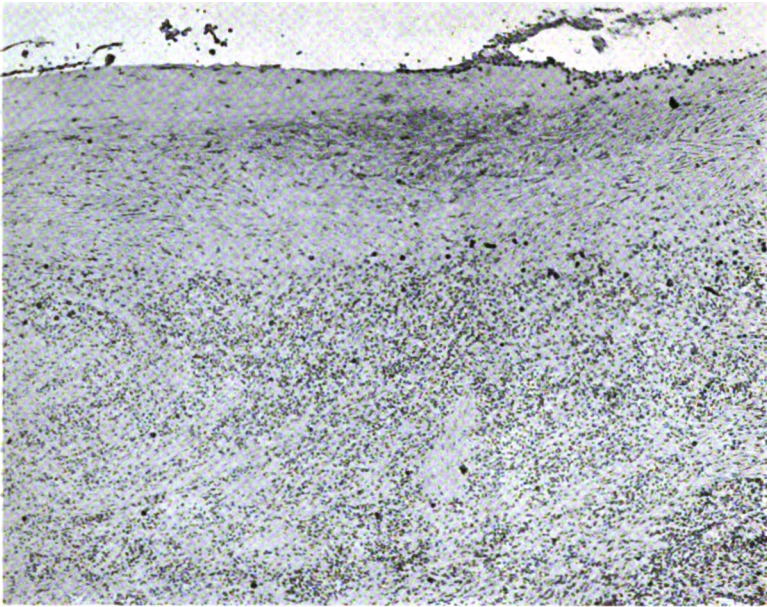
FIG. 5.



Spleen to the left is from a case of splenic thrombosis, the one to the right from a case of hepatic cirrhosis.

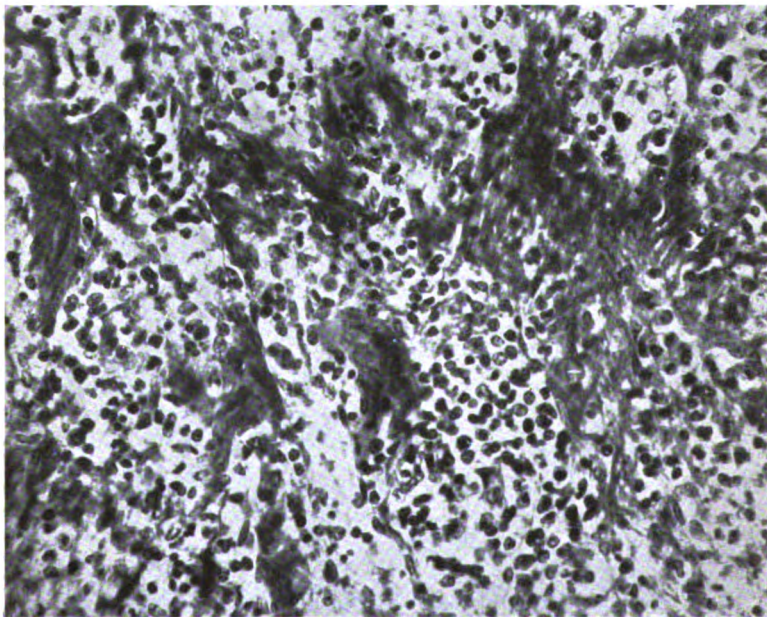


FIG. 6.



Section of spleen in splenic anaemia with thrombosis of splenic vein. Fibroid hyperplasia.

FIG. 7.



Section of spleen. Case of splenic anaemia with thrombosis of splenic vein. High-power view. Increase of stroma.

ciated with splenic enlargement. These cases are reported here to prove the occurrence of acute infections of the portal tract other than syphilis, and that splenic and portal thrombosis may be a sequel or complication of an acute infection, such as pneumonia, or of a more chronic one, such as the case of malignant endocarditis. Moreover, it seems highly probable that such infections of the splenic or portal vein may be the cause of splenic anæmia.

**CASE IV. (Dr. de Nancrede).—**Mrs. H., age, 28; nationality, American; occupation, housewife. Day and hour of death, noon, Jan. 19, 1906. Time of autopsy, Jan. 20, 1906. Clinical diagnosis, empyema, post-pneumonia, sub-mucous fibroid. Family history, negative; measles and whooping-cough before the age of six. Menstruated at 18 years; painful and irregular for first year, then regular until two years ago, when menstruation became painful and irregular. At nineteen had pleurisy on the left side; and has had frequent attacks of quinsy.

Present trouble began early in December, 1905. Sudden attack of severe chill, aching in bones, vomiting, temperature 39.5° C. (103° F.), severe cough, large amount of expectoration for several days. Severe pain in the left side with increasing severe dyspnœa. Sputum was thick and tenacious, never blood-stained. On the fifth and sixth days became better. Left side was dull over the base, and the presence of fluid was suspected. A trocar was introduced in the seventh intercostal space 5 cm. (two inches) from the spinal column, but no fluid was obtained.

Pain in left side with marked tumor in left hypochondrium, continued with the first week in January, when patient entered hospital. On examination a clinical diagnosis of empyema following pneumonia was made and a trocar was passed on January 8, 1906. About 250 c.c. (half a pint) of pus obtained. A streptococcus was found in this fluid. On January 15th the left eighth rib was resected (Dr. de Nancrede) and a large amount of pus obtained from left pleural cavity. Drainage tube introduced, etc. Patient died on the 18th.

The white cell count in this case ran low, 5 to 12,000, in spite of the purulent condition present. Patient was anæmic; red blood-corpuscles, 2,500,000, hæmoglobin about 50 per cent.

#### AUTOPSY PROTOCOL: PROSECTOR: DR. BUTTERFIELD

*External Examination.*—Build: Height, 153 cm. (5 feet), muscles fairly developed. Flabby development of musculature. Graceful build. Superficial veins of thighs, arm and hands dilated. Abdomen slightly above level of ribs. Tumor in left hypochondrium. Visible mucous membranes very pale. Conjunctiva pale, colorless, sclera muddy white. Skin extraordinarily pale, sallow. General œdema. Abdominal fat abundant, yellow. Abdominal muscles dark red. Abdominal cavity contains 300 or 400 c.c. (about 12 fluidounces) turbid yellow-red fluid. Puncture mark 5 cm. (2 inches) below umbilicus, edges of which are green. Rigor mortis, in extremities. Panniculus, fairly abundant. Œdema, marked; inframammary region shows small puncture marks.

Body heat, absent. Hypostasis, slight posteriorly. Surgical dressings about thorax and left chest wall, incision drain, dressing in sixth I.C.S. in anterior axillary line.

**Thorax:** Diaphragm, adhesions between diaphragm and adjacent structure; on left upper border, fourth rib; on right, third I.C.S. Adhesions universal throughout abdominal cavity. Mediastinum, fat abundant. Thymus, no vestige of thymus tissue found. Pleural cavities, right contains 200 c.c. (6 fluidounces) thin, blood-stained fluid; free from adhesions; left almost obliterated by adhesions, most of them easily separated, few were tenacious; left contains between adhesions at base 500 to 600 c.c. (about 1 pint) dark red, turbid fluid, offensive odor. Pericardium, cavity contains a few c.c. (about a fluidrachm) of straw colored fluid; layers are smooth and glistening; no adhesions. Heart, 255 Gm. (9 ounces). Trifle larger than patient's fist. Subpericardial fat abundant. Heart quadrilateral in shape; gives readily. Muscle pale, reddish brown. Foramen ovale closed. Right auricle distended by clotted blood. Right ventricle distended by clotted blood. Walls loose and flabby. Wall right ventricle 3 to 6 mm. ( $\frac{1}{8}$  to  $\frac{1}{4}$  inch). Left auricle contains small amount clotted blood. Wall left ventricle 11 mm. Wall left ventricle 3 to 6 mm. ( $\frac{1}{8}$  to  $\frac{1}{4}$  inch). Valves, tricuspid valves delicate; present no abnormalities. Left auriculoventricular admits 2 fingers. Anterior leaflet of mitral presents few nodules, thickenings along free border; firm, yellowish in color. Mitral valve normal. Coronary patulous. Aortic valves normal except for few fibrous patches. Left lung, small; air-containing; slight emphysema. Lower lobe and lower part upper lobe, crepitation markedly diminished. Firm tags of membrane organizing fibrinous exudate on lower lobe; dark red in less crepitant portion. On section moist. Numerous subpleural petechiæ. Lower lobe airless; sinks in water. Lungs very friable, especially lower lobe of left. Right lung, apex of upper lobe, outer border, emphysematous; slight anthracosis. Lower lobe less crepitant; dark; slight purplish tint. On section, upper lobe gray-white; cut surface slightly moist. Lower lobe, surface moist; slightly blood-tinged fluid; slight œdema and congestion in lower lobe. Base of lung adherent to diaphragm. Bronchi contain small amount of frothy, slightly tenacious fluid. Bronchial glands small and anthracotic on right; enlarged and pigmented on left. Great vessels of thorax; aorta, slight beginning atheroma. Trachea contains a considerable amount of frothy, tenacious material.

**Abdomen:** Adhesions universal throughout abdominal cavity. Omentum, injected; tip adherent to pelvis; very fatty. Position of abdominal organs, lower border of liver 5 cm. (2 inches) below xyphoid. Spleen extends a hand-breadth below level of ribs; greatly enlarged. Spleen: Adherent to diaphragm and liver; adjacent tissues show fibrinopurulent, hemorrhagic exudate, indicating intense inflammation. One-third of spleen tissue is occupied by soft, purulent mass breaking on removal discharging cup full of pus. Rest of spleen shows pus collections of varying size. Between spleen and liver is a walled-off collection of pus. Yellowish, thick. Pus is tinted pink. Splenic vein completely obturated by a partially organized thrombus. Wall of vein greatly thickened, sclerotic, and containing small areas of calcification. Left kidney, perirenal fat and fatty capsule strip easily, leaving smooth surface showing stellate veins. Fat abundant. Pale on section.

Labyrinth pale, yellow-gray hue. Straight vessels contain pale blood. Pyramids pale. Cortex measures 4 mm. ( $\frac{1}{8}$  inch). Pelvis fat abundant. Right kidney similar to left. Bladder, few c.c. (about a fluidrachm) fairly turbid urine. Wall thin. Mucous membranes pale. Mucosa thin and smooth. Duodenum slightly bile-tinged. Stomach contains a few c.c. (about a fluidrachm) turbid grayish-yellow fluid, containing whitish particles; is adherent to under surface of liver at fundus. Small intestine, surface covered with thin slightly tenacious substance. Firm adhesions between omentum and underlying intestines. Mucosa thin, smooth, pale, with exception of area of 40 cm. (25 inches), in which the walls are very thick, friable, and hemorrhagic. Villi œdematous. Tips of valvulæ covered with light, diphthoritic, necrotic material. Appendix long, curled on itself; directed posteriorly and downward; lumen patent throughout. Large intestine, tags of fat on colon. Mucosa smooth and thin. No ulcers in either large or small intestine. Liver, pallor of blood very noticeable. Reaches to left side of diaphragm. Abscess in left lobe. Moderate size. Left lobe relatively large. Surface covered with moderately old fibrinous material, yellowish, brownish-pink, with scattered purplish-red patches. Moderately firm on section. Surface moist. Lobular markings exaggerated. Central vein distended with pale blood. Light yellowish opaque areas about central vein. Drainage tube entered left leaf of the diaphragm seventh I.C.S. One drainage tube inserted under left lobe of liver; the other directed downward into sac formed by parietal peritoneum and omentum. Rib resected to correspond to entrance of tube. Gall-bladder firmly adherent to colon; contains no stone; moderately tense. Portal vein, thrombus extends from splenic vein into portal. Walls thickened. Pancreas, firm; apparently normal. Pancreatic veins contain fresh thrombi. Vessels of abdominal cavity, no thrombi in iliac vessels. Enlarged pedicle. Vagina, walls thin and smooth. Uterus, large submucous polyp protruding from uterine cervix into vagina. Surface injected and hemorrhagic. About neck is tenacious mucoid material. Ovaries, right ovary firm; contains few small cysts. Surface markedly wrinkled.

*Microscopical and Bacteriological Findings.*—Splenic and portal veins, fresh thrombus, slight beginning organization. Walls œdematous, and show active inflammation and recent hemorrhage. Perivascular tissue inflamed. Patches of fibrin outside the vessel walls. Spleen, multiple anæmic infarcts. Infarcts undergoing coagulation necrosis. Considerable fibrin. Large infarct, broken down, purulent, giving rise to abscess. Considerable organization about some of the infarcts. Numerous hemorrhages and dilated blood-spaces. Lymphoid tissue atrophic. Tissue (intra-splenic) showed advanced fibrosis. Few Malpighian bodies. Trabeculæ close together in atlas. Reticulum markedly increased. Colonies of cocci in pus of splenic abscess and in thrombus. Omentum, omental tissue markedly inflamed. Omental vessels contained numerous thrombi. Diaphragm, acute inflammation on peritoneal surface. Muscles showed slight infiltration. Entire omentum showed acute inflammation. Lung, left lung completely atelectatic. Right lung œdematous and congested. Pleura shows recent fibrinous inflammation. Heart, muscle shows cloudy swelling; slight fatty change. Kidneys, anæmic. Epithelium shows cloudy swelling. Slight fatty degeneration. Adrenals showed advanced post-mortem change. Interstitial connective tissue considerably increased. Liver anæmic;

slightly atrophic; slight fatty degeneration; periportal infiltration. Intestine, large red coil of ileum not gangrenous, simply intensely congested and shows advanced degeneration of surface epithelium, probably *post mortem*. Peritoneal surface showed fibrinous inflammation. Fallopian tubes, proliferative chronic salpingitis. Appendix obliterated.

*Pathological Diagnosis.*—Subacute thrombophlebitis of splenic vein; hyperplasia of spleen, abscess of spleen. Streptococcus infection. Infected infarct causing subdiaphragmatic abscess. General peritonitis. Operation wound in left lateral thoracic region; resection seventh rib; perforation of diaphragm by incision and drainage tube. Pyopneumothorax. General anæmia. Atrophy. Anasarca. Atelectasis. Slight pleurisy of left lung. Fibrosis of spleen, pancreas, and adrenal. Chronic salpingitis.

CASE V.—Miss B., student, aged 26 years, entered the medical clinic of the University Hospital (Dr. A. W. Hewlett), November 11, 1909. Father has locomotor ataxia, otherwise family history is negative. At birth patient was a "blue baby." Family physician said there was a congenital leak in a heart valve. Patient had measles, whooping-cough, and chicken-pox as a child, and "typhoid fever" at nine years of age. During the attack of typhoid fever the mother said a grating sound could be heard with each heart beat. Was hearty as a girl with the exception that she could never run around with the other children because she so easily became tired and out of breath. Gets out of breath on going up stairs. Has never had rheumatism or sore throat. Examination made in the University gymnasium in October, 1908, showed the presence of a systolic murmur, loudest over the tricuspid area, and not transmitted into axilla. Heart intermittent. A record made in April, 1909, stated that beat was stronger, apex displaced to nipple-line, and murmur transmitted to axilla.

Present trouble began suddenly December 10th, with chill, fever, headache and faintness. Temperature 40° C. (104° F.) when admitted. Examination by Dr. Hewlett records a prominent præcordium, diffuse heaving impulse, loud blowing systolic murmur at apex transmitted to axilla, disappearing over pulmonic and tricuspid areas. Both second sounds loud, the shock of pulmonic second felt. No diastolic murmur. Slight irregularity apparently due to occasional extra systoles. Splenic dulness somewhat increased, spleen not palpable, although it had been palpable the day preceding (Dr. Gordon).

During the week following admission patient had constant fever, 40° to 41° C. (104 to 106° F.), repeated chills, severe frontal headache. Developed petechial spots over abdomen. Blood cultures showed the presence of streptococci. Injections of a polyvalent antistreptococcus serum given at intervals. Condition gradually became worse with slight intervals of apparent improvement. Purpura increased; slight icterus developed, and patient died January 4, 1910, fifty-two days after admission.

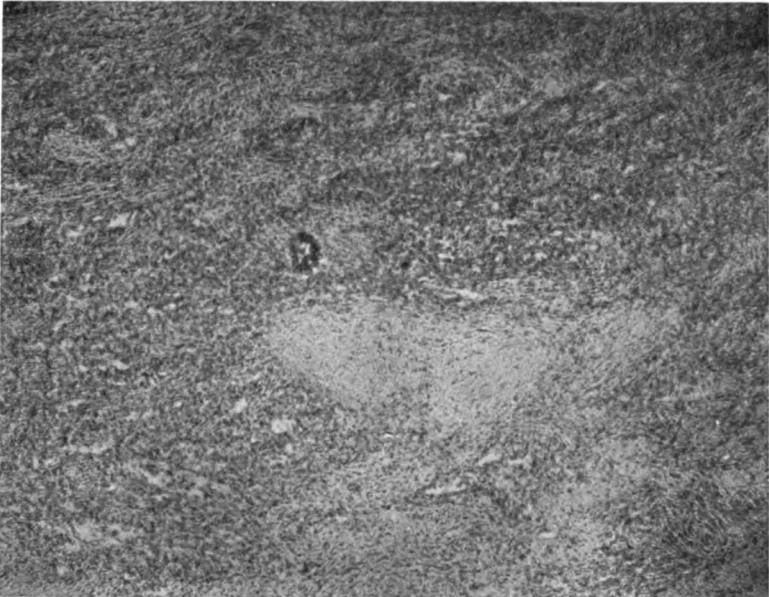
The spleen was never palpable after the first day in the hospital, although at one time the lower edge of the liver was distinctly felt. Abdomen noted as full, but not tender. No especial symptoms at any time referable to splenic region or abdomen. The blood examinations showed a leucocytosis running from 13,000 to 22,000, red blood-cells 2,690,000, hæmoglobin 45 to 58 per cent.

**FIG. 8.**



**Section of spleen from case of splenic anaemia with thrombosis of splenic vein. Thickened and hyaline capsules.**

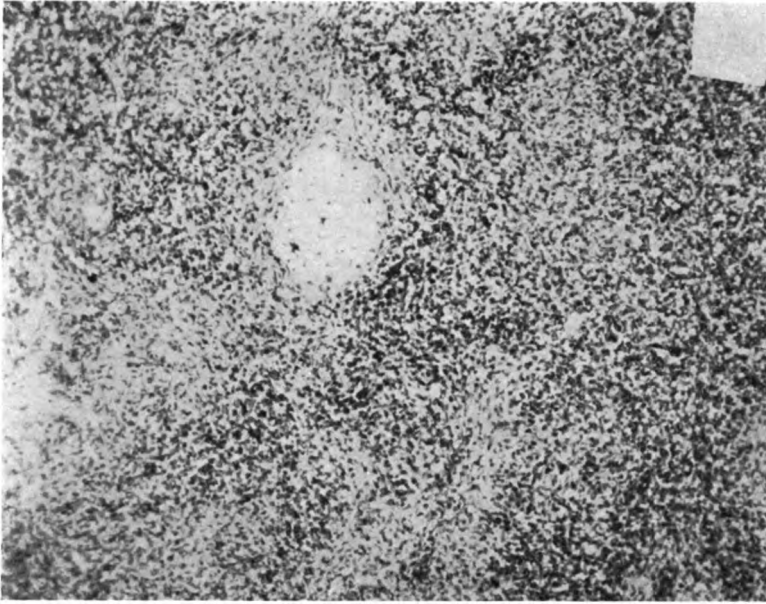
**FIG. 9.**



**Section of spleen from case of recent thrombosis of splenic vein. Increase of stroma.**



**FIG. 10.**



**Section of spleen from case of recent thrombosis of splenic veins. Increase of stroma.**

**FIG. 11.**



**Section of spleen from case of recent thrombosis of splenic vein. Edge of infarct.**

**AUTOPSY PROTOCOL, No. 9, 1900-10.**—Miss B.; age, 26; nationality, American; status, single; occupation, student. Day and hour of death, Jan. 4, 1910, 3 A.M. Time of autopsy, Jan. 4, 1910, 9 A.M. Clinical diagnosis, malignant endocarditis; streptococcic septicæmia; mitral insufficiency; infarction of brain and kidney; septic jaundice. Pathological diagnosis, subacute and chronic vegetative endocarditis on mitral; mitral stenosis and insufficiency; cardiac hypertrophy and dilatation; purulent thrombosis in left auricle; polypoid thrombi in both left and right ventricles; acute purulent myocarditis; embolic miliary abscesses in heart wall; subacute pericarditis. Anæmic infarction of brain, spleen, and kidneys. Hemorrhagic infarction of the lungs. Purulent induced thrombi and purulent emboli in splenic and renal arteries. Septic pyæmia. Marked nutmeg liver with beginning miliary abscesses. Subacute parenchymatous degenerative nephritis. Aneurism of splenic artery. Subacute thrombophlebitis of splenic vein and radicles of mesenteric veins. Acute metastatic thyroiditis. Congestion and parenchymatous degeneration of all organs; congenital syphilis.

**External examination:** Build, moderately slender; slight prominence of stomach region, cardiac area, and left, third, fourth, and fifth costal articulations. Abdomen, level of ribs. Surgical wounds, three transfusion marks; two under left breast, one under right breast. Skin, sallow, yellowish cast over thorax, sclera clear, small blotches of discoloration over thorax and abdomen. Small petechiæ over abdomen, thighs, etc. Small hemorrhage over left gluteal region. Mucous membranes pale. Rigor mortis throughout. Oedema, trace throughout. Body heat, slight warmth in trunk. Hypostasis, marked purplish-red.

**Head:** Skull-cap, thin; unusually large pacchionian erosions. Dura, adherent but not thickened. Intradural pressure increased. Longitudinal sinus, negative. Meningeal vessels, pial vessels everywhere injected. (Dura adherent to meninges over larger area in left parietal lobe. Shows marked discoloration and softening. Arachnoid clouded all over, especially on left. Subarachnoid fluid increased. (Edema.) Inner meninges, left markedly clouded, adherent over parietal lobe; right, cloudy. Cerebrum, surface markedly oedematous. On section, moist and shining; numerous bleeding points. (Edema and congestion. Area of infarction involving gray matter. Beginning abscess size of pin-head. Cortex discolored and softened over parietal lobe. Softening recent. Ventricles, dilated; corpus callosum arching upward, large amount of fluid escaped; third, dilated; fourth, negative. Cerebellum, both lobes moist and oedematous. Pons, small hemorrhage size of pin-head on right. (Edema and congestion. Medulla, oedema and congestion. Hypophysis, negative. Basal sinuses, distended; small amount of jelly clot.

**Abdominal cavity:** Freed fluid; light yellow, slightly turbid. Flakes of albumin and fibrin. Panniculus, fair amount; moist, shining; light orange-yellow. Large amount of subcutaneous fat in median line. Omentum, moderately rich in fat; extends into pelvis. Omentum, cæcum and transverse colon moderately distended. Sigmoid slightly, and small intestine collapsed.

**Position of abdominal organs:** Liver enormously extended on left. Four fingers below umbilicus in left parasternal line; elongated. Round ligament of liver in median line. Gall-bladder small, moderately distended with bile,



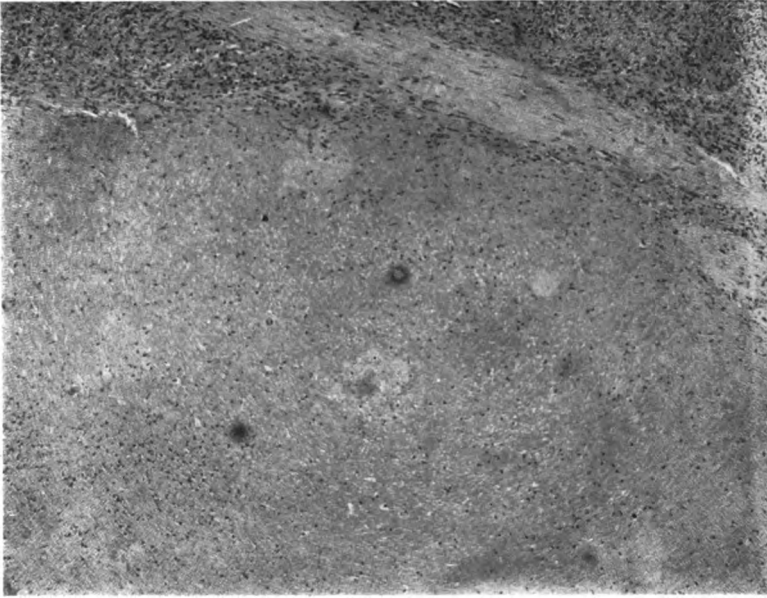
in right parasternal line. Spleen covered by greatly enlarged left lobe of liver. Position of diaphragm: on right, fourth rib; on left, fifth rib. Mammæ, right contains dilated gland spaces containing turbid fluid. Costal cartilages, soft. Sternum, negative.

**Thoracic cavity:** Thorax when stripped almost pigeon breast in contour. Interspaces 2 fingers in width at first, second, third, and fourth. Moderately corset-shaped below. Position of thoracic organs: apex of heart behind fifth rib at nipple line. Anterior mediastinum, fat in abundance; slight œdema and injected. Thymus, small amount of fat. Distinct remains of gland, particularly the right lobe. Pericardium, sac dilated; moderately lax; contains 90 c.c. (3 fluidounces) amber fluid containing fibrin and albumin. Heart, right auricle and ventricle enormously dilated. Cone-shaped apex formed by left ventricle. Cardiac veins, greatly congested. Petchial hemorrhages under epicardium. Subepicardial fat increased; œdematous. Foramen ovale closed. Right heart, polypoid white thrombi between muscle trabeculæ, showing simple softening. Left heart, dilated; muscles flattened; polypoid thrombi behind muscle columns near apex; muscle light colored, soft, and cloudy; fatty shine; chordæ tendinæ very slightly thickened. Cardiac orifices and valves: aortic orifices adequate. Mitral orifice nearly blocked by thrombus which projects from posterior wall of left auricle. Proximal edges of flaps covered with vegetations in many stages of organization; confined mostly to auricular aspect; orifice much wider than normal; tricuspid admits 4 fingers; negative. Left lung, free throughout. 100 c.c. (3 fluidounces) slightly turbid fluid in left pleural sac. Moderately voluminous. Anthracosis well marked. No airless areas. Moderate congestion. Fresh hemorrhagic infarct underneath the pleura. Right lung, adherent posteriorly; an old adhesion which comes off with great difficulty. Pleural sac contains 100 c.c. (3 fluidounces) fluid. Numerous old adhesions. More œdematous than left. No airless areas. Abundant foamy fluid on pressure. Bronchial glands, small; pigmented. Two healing tubercles. Pulmonary vessels, aorta greatly dilated, admits 2 fingers with ease; flaps negative. Great vessels of thorax, inferior vena cava enormously dilated; contains thin watery blood with small amount of jelly clot. Aorta dilated; admits 2 fingers.

**Pharynx,** mucosa swollen; thickened; chronic pharyngitis. **Larynx,** negative. Trachea, small hemorrhage just below vocal cords. Cervical portion of œsophagus, negative. Thyroid somewhat enlarged, rather small amount of colloid. Parathyroids, negative. Submaxillary gland thickened; swollen.

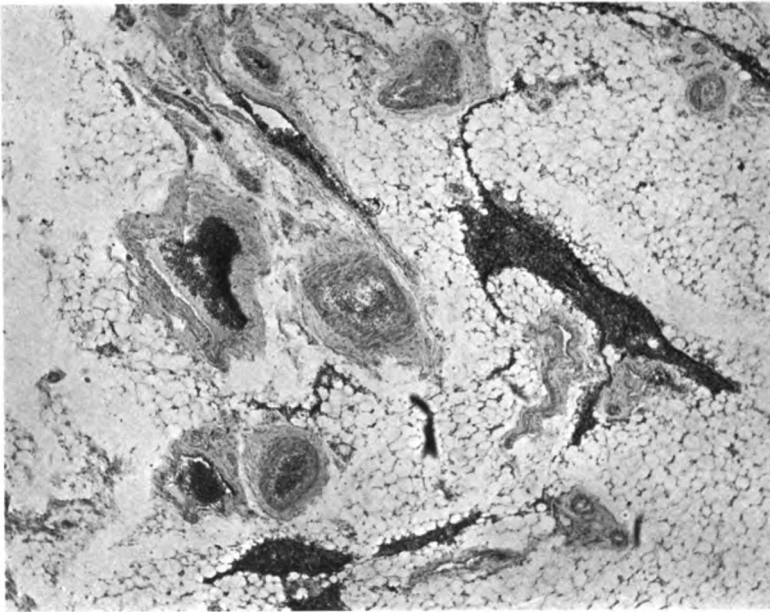
Spleen, adherent to diaphragm by fairly dense adhesions; many infarcts; multiple anœmic infarcts in all stages; aneurismal dilatation of splenic artery near hilum; thrombus in splenic vein, at hilum of spleen, size of horse-chestnut. Splenic vein thrombosed for half its distance. Lumen completely obturated in places. Left adrenal, post-mortem change in medulla; œdema of surrounding fat. Left kidney and ureter, fatty capsule well developed, œdematous; fibrous capsule strips easily. Fetal lobulations somewhat preserved. Surface mottled; purple and grayish; cortex cloudy; outlines between medullary rays and labyrinth indistinct; fatty shine; pelvis negative. On anterior wall one small anœmic infarct. Acute degenerative parenchymatous nephritis. Right adrenal, negative. Right kidney and ureter, same as left, except no infarcts seen. Large intestine, lower portion

**FIG 12.**



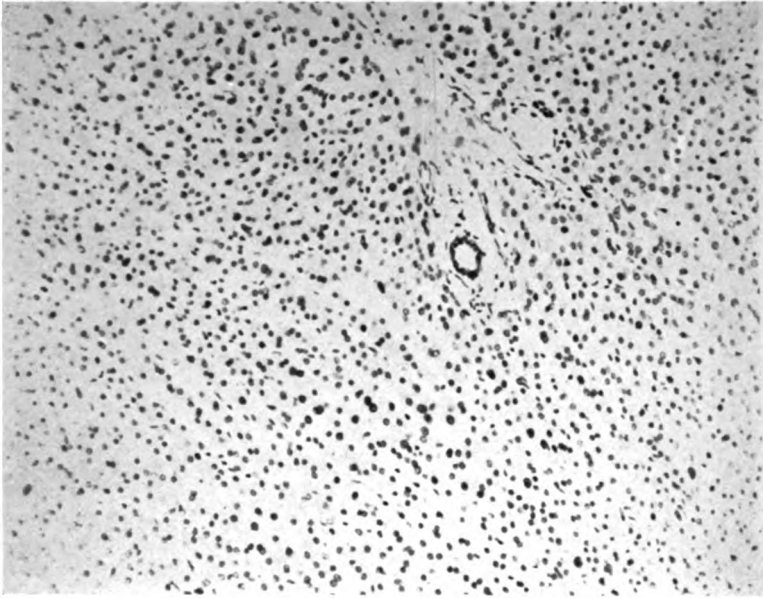
**Section of spleen from case of recent thrombosis of splenic vein. Edge of infarct.**

**FIG. 13.**



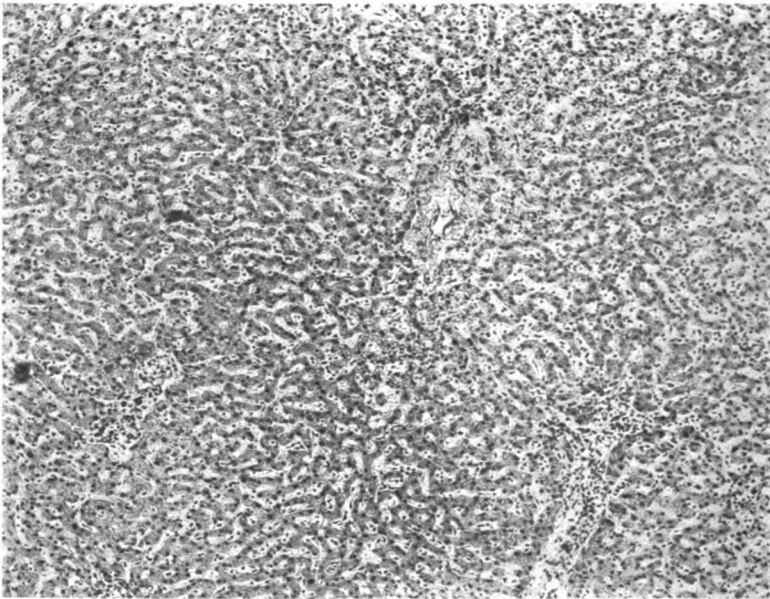
**Portal radicles in mesentery. Case of splenic anaemia with thrombosis of splenic vein.  
Small veins dilated and showing thickened walls.**

FIG. 14.



Liver from early stage of splenic anæmia with thrombosis of portal and splenic veins. Atrophy. No increase of stroma.

FIG. 15.



Liver from later stage of splenic anæmia with thrombosis of splenic vein. Atrophy. Some increase of stroma.

shows marked mucous catarrh. Small intestine, mucosa oedematous and congested throughout. Stomach contains brownish-gray, finely divided, sour-smelling material. Mucosa shows beginning post-mortem change. Numerous petechial hemorrhages. Pancreas, fairly firm; congested. Liver, corset liver; thickening of capsule corresponding to edge of ribs; nutmeg; chronic passive congestion. Abdominal aorta: Small; intima shows inhibition of hæmoglobin and lines and patches of marked fatty change. Hæmolymp-nodes: retroperitoneal hæmolymp-nodes hyperplastic mesentery. Radicles of mesenteric veins sclerotic, some showing active phlebitis and thrombophlebitis.

Bladder much thickened and injected; contains a small amount of purulent exudate. Vagina, patent, small amount of purulent exudate. Uterus, negative. Ovaries normal.

*Microscopical and Bacteriological Findings.*—Heart, interstitial myocarditis. Embolic miliary abscesses throughout the heart wall. Subacute pericarditis. Mixed thrombus of left auricle with beginning organization and purulent softening. Subacute vegetations on mitral. Sections of left auricle show early purulent myocarditis. Purulent thrombi in left and right ventricle. Colonies of cocci. Lung, marked oedema and hemorrhage. Recent hemorrhagic infarction. Numerous recent purulent emboli. Beginning miliary abscesses. Brain, area of beginning softening. Anæmic infarction with small beginning embolic abscesses. Thyroid, acute metastatic thyroiditis. Spleen, all stages of anæmic infarction. Emboli and thrombi of splenic arteries and veins, showing all stages of organization, suppuration, etc. Numerous beginning abscesses containing colonies of cocci. Marked atrophy of lymphoid tissue. Increase of reticulum. Splenic artery, just outside the spleen an aneurism the size of a black walnut containing a mixed thrombus with beginning organization. Tissues about artery and vein show repeated hemorrhages and hæmosiderosis. Large clumps of hæmosiderin found in the wall of the artery and in the thrombus. Splenic vein, subacute phlebitis; organizing thrombus. Kidneys, subacute, degenerative nephritis; hyalin and blood casts; marked cloudy swelling and congestion. Mesenteric vessels, small radicles of portal show thrombophlebitis. Pancreas, cloudy swelling, oedema, and congestion. Liver, nutmeg with beginning miliary abscesses in large numbers; atrophy. All other organs show congestion, oedema and purulent emboli with miliary abscesses.

#### EXPERIMENTAL

A number of series of experiments were carried out to ascertain the effects upon the spleen of partial and complete ligation of the splenic and portal veins. Rabbits and dogs were used for this work. In the case of both animals it was found extremely difficult to shut off completely the venous flow from the spleen because of the number of veins and the rapidity with which a collateral venous circulation was established. In no case was it possible to produce a permanent complete obstruction to the venous outflow of the spleen. In the animals allowed to live longest after the operation, a partial

restoration of the venous circulation was always found, chiefly by the formation of collateral branches in the gastrosplenic omentum.

The animals were operated upon under the proper conditions of anæsthesia, etc. They stood the operation well, and when not infected showed no signs of disturbed metabolism. The tendency to increase of fat was not shown in the rabbit as in the case of the dogs killed by chloroform at varying times after the operation, from twenty-four hours to eighteen months. A complete autopsy was made in each case with microscopical examination of all the organs. Especial attention was given to the changes in the spleen, liver, and the blood-vessels of the portal system. The red and white cells were counted at varying intervals. A typical series of experiments follows, one for each animal used.

#### SERIES I.

1. *Gray Rabbit*.—All of the visible splenic veins were ligated, using a small curved needle and silk thread. The spleen immediately increased in size, became darker in color and firmer. Animal killed in 24 hours. Spleen was about one-third larger than normal, firm, dark, bluish-red in color. No changes in other organs microscopically. The spleen showed a marked passive congestion with some pigmentation. Other organs negative.

2. *Belgian Hare*.—Ligation of splenic veins. Immediate passive congestion of spleen as shown by enlargement of the organ and by its deeper color and firmer consistency. Animal killed one week later. Spleen enlarged and firm, and darker in color. Microscopical examination showed a passive congestion of the organ, with increased pigmentation. No proliferation of connective tissue or endothelium was seen. Other organs negative.

3. *White Rabbit*.—Ligation of splenic veins with immediate enlargement of organ. Lower two-thirds of spleen enlarged about one-half, firm and dark brownish-red. Upper third slightly larger than normal, lighter and firmer. Microscopical examination showed chronic passive congestion of entire spleen, more marked in the lower two-thirds. Atrophy of lymphoid tissue with relative increase of stroma in the upper third and excessive pigmentation. No changes in portal vein, mesenteric veins, or liver.

4. *Gray Rabbit*.—Ligation of splenic veins with resulting slight increase in the size of the organ. Animal killed three months after. Spleen smaller than normal, firmer, and light brown in color. Microscopical examination showed marked atrophy of the lymphoid tissue and relative increase of the stroma, with excessive pigmentation (hæmosiderin). Portal and mesenteric veins and liver showed no changes.

5. *Belgian Hare*.—Splenic veins ligated. Animal killed six months later. Spleen irregular with deep constrictions separating the organ into a number of islands, each one being connected with a vein of good size in the gastrosplenic omentum. Microscopical examination showed marked atrophy of

**FIG. 16.**



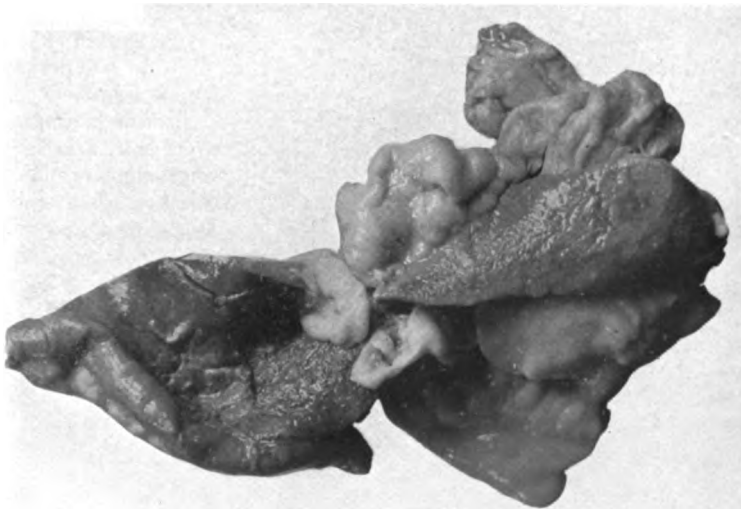
Spleen of rabbit, six months after attempted ligation of splenic veins. Chronic passive congestion of lower half, atrophy of upper portion.

**FIG. 17.**



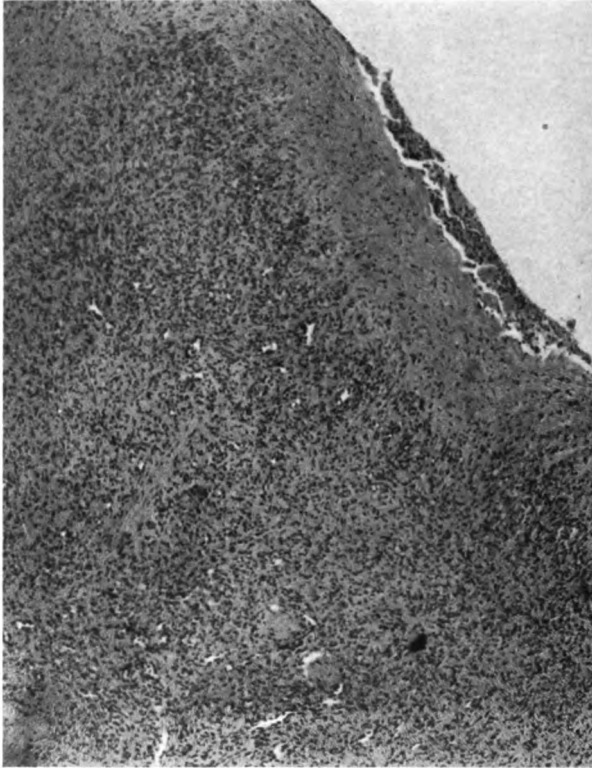
Spleen of rabbit three months after attempted ligation of splenic veins. Spleen small, very firm, and lighter in color.

**FIG. 18.**



Spleen of dog, eighteen months after ligation of splenic veins. Atrophy of central portion of organ.

**FIG. 19.**



Section of spleen of dog eighteen months after ligation of splenic veins. Thickened capsule and increase of stroma with atrophy of lymphoid tissue.

splenic tissue at the constricted areas, with excessive pigmentation. The larger areas of preserved splenic tissue were congested and also pigmented. No absolute increase of stroma. Blood-vessels and liver showed no changes.

#### SERIES II.

1. *Dog*.—Ligation of splenic veins. Slight immediate enlargement of the spleen. Examined one week after ligation. Spleen swollen, edges rounded, consistency increased, color dark bluish-red. Microscopically, the spleen showed a condition of passive congestion. Other organs negative.

2. *Dog*.—Ligation of splenic veins. Immediate enlargement of spleen, the organ increasing in size about one-sixth, becoming firm and dark purple in color. Animal examined two weeks. Spleen still about one-sixth larger than normal, firm and dark in color. Dilated veins in gastrosplenic omentum. Microscopically the organ showed passive congestion with excessive pigmentation.

3. *Dog*.—Splenic veins ligated. Spleen immediately became larger, firmer and darker in color. Examined three weeks later. Spleen large, its edges rounded and firm, color dark brownish red. Beginning development of venous collaterals along splenic artery and in the gastrosplenic omentum. On microscopical examination the spleen showed a chronic passive congestion with lymphoid atrophy and relative increase of the stroma, with excessive pigmentation (hæmosiderosis). The ligated splenic veins contained organized thrombi. Numerous dilated venules along branches of the splenic artery and in gastrosplenic omentum. Other organs negative.

4. *Dog*.—Splenic veins ligated near the hilum. Immediate enlargement of the spleen, about one-tenth in size. Edges swollen, firm, color dark purple. One month later spleen was enlarged, dark bluish-red, firm, edges rounded. Veins at hilum partly obliterated by organized thrombi; a few widely dilated veins along the divisions of the splenic artery and in the gastrosplenic omentum. Microscopically, chronic passive splenic congestion, with lymphoid atrophy and relative increase of stroma. Hæmosiderin in abundance. Organs negative.

5. *Dog*.—Ligation of divisions of splenic vein near the hilum. Immediate enlargement of spleen, the organ becoming firmer and darker. Animal autopsied three months later. Spleen irregular in size and form, on the whole somewhat smaller than normal, brownish-blue in color and much firmer than normal. Development of small collateral veins in omentum and along branches of splenic artery. On microscopical examination the spleen showed irregular areas of marked atrophy of the lymphoid tissue with relative increase of the stroma, and excessive hæmosiderosis. The remaining portions of the organ showed chronic passive congestion. In the hilum a number of veins obliterated by organized thrombi were present and these corresponded to the atrophic portions of the organ. The animal was very fat and the liver showed marked fatty infiltration. No cirrhosis. The prevertebral lymph-nodes and hæmolymph-nodes were slightly hyperplastic. Other organs negative.

6. *Dog*.—Splenic vein ligated near portal. Immediate enlargement of spleen, but to a less degree than when the ligation was made near the hilum. Animal autopsied a year later. Body very fat. Spleen small and atrophic, deep-brown in color, consistence firm. Collateral circulation in gastrosplenic



omentum. Prevertebral lymphatic and hæmolymp-nodes hyperplastic. Fatty liver; no cirrhotic changes. Other organs negative.

7. *Dog*.—Splenic veins ligated near spleen. Immediate enlargement; organ became very hard, dark, and swollen. Edges rounded; capsule stretched. Autopsied eighteen months later. Animal very fat. Spleen small, irregular in shape; about its middle completely separated into two lobes or islands of splenic tissue, each having separate veins running into the gastrosplenic omentum and anastomosing with veins from the stomach. Both lobes firm and dark-brown in color. On section marked atrophy of the lymphoid tissue with fibroid change in the stroma was found in the portions of the organ bordering upon the fissure separating the two islands. At this point small microscopical islands of splenic tissue were found in the adipose tissue, and veins obliterated by organized thrombi occurred at this point also. The absence of such a fissure in the spleen at the time of operation shows conclusively that it developed after the ligation of the splenic veins as a result of the nearly or quite complete obstruction to the venous outflow from that portion of the spleen. The remaining portions of the organ showed a chronic passive congestion with excessive hæmosiderosis, lymphoid atrophy and relative increase of the stroma. The liver was very fatty, but showed no cirrhotic changes. The retroperitoneal hæmolymp-nodes were hyperplastic.

#### SUMMARY OF EXPERIMENTAL WORK

Other control experiments confirmed the above results. Some variance was found in regard to the hyperplasia of the hæmolymp-nodes. This did not always occur, especially in marasmic dogs kept under poor conditions. Hyperplasia was most marked in well-nourished dogs. Regeneration of the spleen was not seen. Summing up the results, they were practically the same in both rabbits and dogs, although more marked in the latter animal.

The changes in the blood were slight. A slight or moderate anæmia followed the operations in all cases. The hæmoglobin was usually reduced to a greater degree than the red blood-cells. No lasting changes in the number or type of the white cells were noted. A slight leucocytosis usually followed the operation; the leucocytes then gradually fell, to rise again slowly. The differential counts showed no constant changes in preparations, but there was first a decrease in lymphocytes (lymphopænia), later an increase. The effects of the ligation of the splenic veins at varying distances from the spleen may be given briefly as follows:

1. Ligation of the splenic veins or vein caused an immediate passive congestion of the organ, with increase in size and consistency and deepening of color. This change was the more marked

the nearer the ligation to the hilum and the more complete the ligation of the veins in the hilum. It was practically impossible to ligate all the veins coming from the spleen, hence some collateral circulation developed in all cases.

2. In dogs and rabbits examined at intervals of from one day to six weeks the spleen was found in a state of passive congestion, enlarged, firm, and dark in color. The maximum enlargement occurred between the fourth and sixth week.

3. After three months the spleen was found to be swollen, paler and firmer, and more irregular in shape. Secondary atrophy of the lymphoid tissue without hyperplasia of the stroma was present.

4. After one year the spleen usually was atrophic as a whole or irregularly so, often cut up into small islands of splenic tissue, each of which possessed separate veins running to the gastro-splenic omentum and anastomosing with veins from the stomach. At the fissures more or less complete atrophy of the splenic tissue was found.

5. No marked evidences of proliferation of the splenic stroma or endothelium were found. Some increase no doubt occurred, but for the greater part there was only a relative increase in the reticulum.

6. Moderate hyperplasia of the hæmolymp-nodes took place. It will be remembered that such a hyperplasia of the hæmolymp-nodes has been noted in splenic anæmia (Warthin, Sanford and Dolley). This must be interpreted as the result of a disturbance of the splenic function, inasmuch as such a hyperplasia occurs also after splenectomy. The excessive pigmentation must also be similarly interpreted as due to disturbed splenic function.

7. No notable changes were found in the liver. If any change occurred it was a slight decrease in size.

From the above experimental work it is evident that the splenic hyperplasia found in man in cases of portal and splenic obstruction due to thrombosis or compression cannot be produced in the rabbit or dog by ligation of the splenic veins. When this operation is performed in these animals, the spleen becomes congested and larger, but there follows an atrophy of the organ, more or less marked, without a fibroid hyperplasia. This cannot, however, be taken as an argument against the production of a fibroid hyperplasia in the human spleen as a result of splenic or portal obstruction. There is certainly a greater tendency on the part of the human spleen to

undergo fibroid hyperplasia from passive congestion than there is in the case of the spleen of the animals used. Moreover, as pointed out above, the collateral venous circulation is, in the case of these animals, very quickly set up, and it is practically impossible to produce an absolute venous stasis in their spleens.

The results of experimental ligation of the portal and splenic veins, as recorded in the literature, vary greatly. Moos, in 1859, ligated the portal vein in frogs and rabbits. After fifteen days the liver was distinctly smaller, and after ninety days very small, pale, firm and showed an increase in connective-tissue. Solowieff (*Virchow's Arch.*, Bd. 62) ligated the portal vein in dogs, and found that after a complete sudden closure of the vessel the animals died within four to twenty-two hours. He then tried alternately ligation of the superior mesenteric vein and the splenic vein, finding that the animals then lived weeks and months. In the animals living longest the livers were small, hard, and anæmic; and showed microscopically the features of an interstitial cirrhosis.

Foa and Salvioli repeated this experiment and found that the results varied with the animal used and the place of ligation. Cohnheim and Litten (*Virchow's Arch.*, Bd. 67) arrived at very different conclusions from experiments made by them, and declared that the complete shutting-off of the portal blood from the liver does not at all affect the integrity of that organ.

The older investigators concerned themselves with the effect upon the liver of portal ligation. My experiments were directed especially towards ascertaining the effects of such a procedure upon the spleen, and I have been unable to find in the literature any records of experimental work bearing upon this point.

As to the effects upon the human liver of portal obstruction it appears certain from the reported cases that atrophy of the liver is nearly a constant finding in cases of portal obstruction, and that in the more marked or older cases a condition of cirrhosis develops. The cases of portal stasis from compression of the portal vein from the outside of the vessel shows conclusively that simple portal obstruction does produce changes in the liver as well as in the spleen.

#### SUMMARY AND CONCLUSIONS

From the cases reported in the literature (Banti, Dock and Warthin, Lossen, Sanford and Dolley, *et al.*) it is clear that in man

the complex of splenic anæmia and Banti's disease, as formulated by Osler, is, in certain cases, if not in all, the result of an obstruction to the splenic and portal circulation, particularly the former, and that this obstruction is most commonly the result of an old thrombophlebitis of the portal and its radicles. Nevertheless the symptom-complex can be produced by compression of portal or splenic vein from without, torsion of splenic vein, dislocation of spleen, etc. We must, therefore, group such portal or splenic obstructions with splenic anæmia or Banti's disease, and we cannot regard these conditions as individual disease-entities, but only as symptom-complexes and pathological complexes due to various causes. Cases of so-called splenic anæmia with neoplastic changes in the spleen fall into another group and are not considered here.

The essentials of the complex of portal and splenic obstruction are large splenic tumor, small liver, hæmatemesis, sudden hemorrhages from mouth and anus, ascites relieved by hemorrhage, splenic tumor, often diminished after hemorrhage, intermittent character of symptoms due to progressive thrombosis with canalization, rarely icterus, very acute or chronic course, secondary anæmia, and usually leucopænia. The writer would class all the old cases of portal and splenic thrombosis, splenic tumor and hæmatemesis with the modern cases of splenic anæmia and Banti's disease, and *vice-versa* all the cases of the latter with those of the former. They all represent the same thing.

In regard to the main symptoms of the complex, the secondary anæmia is the result of hemorrhage and disturbed splenic function; the leucopænia is the result of the anæmia (this condition is common to the majority of severe anæmias), the disturbed splenic function and the cachexia resulting from disturbance of gastro-intestinal digestion. The hyperplasia of hæmolymp-nodes sometimes found is an evidence of compensation for lost splenic function. Likewise, the numerous red-cell-containing phagocytes seen in the spleen in some cases show the disturbance of the hæmolytic function of the spleen. The intermittent character of the process, as mentioned above, is dependent upon the changes in the lumen of the obturated or stenosed vein.

As to the liver changes in the early and middle stages of uncomplicated cases (absence of syphilis, alcoholism, etc.), these consist

of anæmia and atrophy of the liver resulting from the loss of the portal circulation. To such an anæmic atrophy and the disturbances of function of the liver-cells a later cirrhosis of atrophic, or more likely interstitial type, could logically follow, adding to the symptom-complex (Banti's). There is nothing specific about the liver changes, no more than of the splenic. The latter organ, however, bears the brunt of the stasis usually, the origin of the thrombophlebitis being most frequently in the splenic or in the portal, extending thence usually into the splenic. The spleen in such cases has not the benefit of the collateral circulation set up by the mesenteric branches of the portal in ordinary hepatic cirrhosis; and the gastrosplenic anastomosis thereby becomes extremely large, in many cases enormous. The extreme length and tortuosity of the splenic veins in some cases suggests an actual increase in length as well as in diameter.

Of all the factors of the symptom-complex the only one that might be the expression of some specific general or local intoxication is the phlebosclerosis of the portal trunks and radicles. The two cases of recent thrombosis of the splenic vein occurring in pneumonia and malignant endocarditis show that this phlebosclerosis can result from a relatively recent infection of the portal vein and its radicles. The whole pathological picture points to an *infective thrombophlebitis of portal or splenic veins as the essential feature of all these cases, no matter under what head reported (splenic or portal thrombosis, splenic anæmia, or Banti's disease)*.

The symptoms of vague abdominal pain and fever, so frequently met with in the earlier stages of these conditions, the relapses, intermittent character, the fact that they often follow acute infections, particularly typhoid, "malaria," pneumonia, etc., all point to a local chronic infection. The etiology of this infective thrombophlebitis is certainly not uniform. Syphilis is probably the most frequent cause, and the evidences of this disease may be localized in this tract alone. Secondary or primary cryptogenic pyogenic infections (streptococcus, as in Case IV and V, above), may explain a large per cent. of the cases, and the symptom-complex may be a sequel or complication of a number of the infections.

That a relatively large number of cases of splenic anæmia have been reported without any mention of portal or splenic obstruction

cannot at this time be taken as an argument in favor of the entity of splenic anæmia. It is most likely that the portal and splenic veins were not thoroughly examined; they are not in the usual autopsy. Until it has been definitely shown that the splenic anæmia or Banti's complex can exist without any evidence of obstruction in portal or splenic vein then my conclusion must hold that the complex is no disease-entity. Should such cases be found, then this criterion must fall to the ground in their case, and a new grouping made. *Until this is done, splenic anæmia and Banti's disease must be regarded as co-ordinated symptom-complexes and not individual disease-entities.*

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# Ophthalmology

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## REFRACTION BY THE GENERAL PRACTITIONER

BY WILLIAM ZENTMAYER, M.D.

PHILADELPHIA

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IN view of the resolution passed by the Section on Ophthalmology of the American Medical Association that "every general practitioner should have the training in ophthalmology which will enable him to manage infectious diseases of the eye and its refractive defects" the EDITOR has asked for a series of articles upon the subject of refraction which will instruct those already in practice how to refract.

The branch of the science of ophthalmology known as refraction being based upon optics, it is absolutely necessary, for even a superficial knowledge of the subject, that the elementary properties of lenses and the way refraction takes place within the eye, be understood. If the testing of eyes for glasses is to be undertaken by the general practitioner it is necessary not only that he be taught the manner of doing this but that he be as fully informed when not to do it. One of the strongest indictments that can be brought against the many "pathies" that have sprung up is that those who practise them do not understand the limitations of their field, and work injury not so much directly as indirectly by withholding from the sufferer competent advice at a time when aid might yet be rendered. This criticism applies particularly to those who without knowledge of disease processes undertake the prescription of glasses, and little will be gained if this work be now undertaken by a physician if he be fully as ignorant of diseases of the eye as the tradesman.

It would be beyond the scope of this article to undertake a description of even the commoner affections of the eye. All that can be done is to point out the indications for glasses and such



conditions and symptoms as indicate the presence of changes other than an anomaly of refraction.

When a ray of light passes from one transparent medium to another of different density it is refracted or bent from its

FIG. 1.

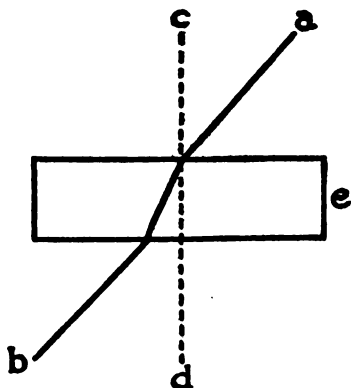
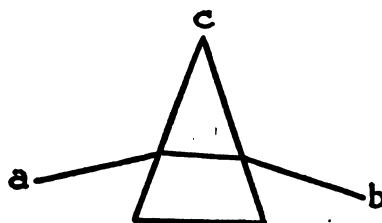
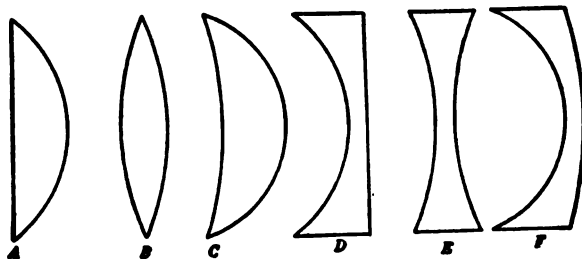


FIG. 2.



direction (Fig. 1, *ab*) unless it meets the surface of the second medium perpendicularly, in which case it passes on without altering its course (*cd*). If the sides of the refracting medium are not parallel, the ray will in a like manner be bent from its course. In a prism, which is a wedge-shaped transparent body, the rays of light are bent towards its base (Fig. 2, *ab*). A lens is a transparent body

FIG. 3.



with at least one curved surface. According to the character of its two surfaces, the lens is designated as: (a) plano-convex, one side the segment of a sphere the other side plane; (b) bi-convex, both surfaces segments of a sphere; (c) concavo-convex, a combination of a convex and a concave surface, the convex surface

being the stronger; (*d*) plano-concave, one side concave the other plane; (*e*) bi-concave, both surfaces concave; (*f*) concavo-convex, a combination of a concave and convex surface, the concave being the stronger (Fig. 3). All of the above forms are designated as spherical lenses. A cylindrical lens is one which is a segment of a cylinder, its surface is plane (linear) in one axis and spherical (circular) with a convexity or a concavity in the axis at right

FIG. 4.

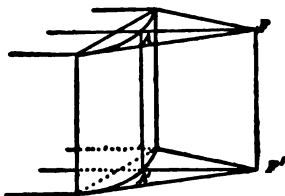
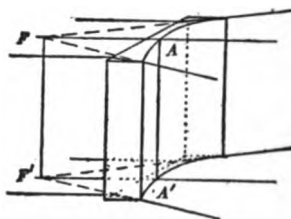


FIG. 5.



angles to the first (Figs. 4 and 5). A sphero-cylindrical lens is one with a spherical surface upon one side and a cylindrical surface on the reverse. A toric lens is one having a surface of two dissimilar curvatures at right angles to each other.

The convex lenses may be looked upon as composed of two prisms with their bases together at the centre of the lense (Fig. 6), while the concave may be considered to be composed of two prisms

FIG. 6.

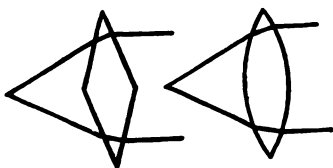
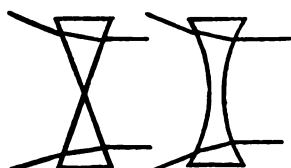


FIG. 7.



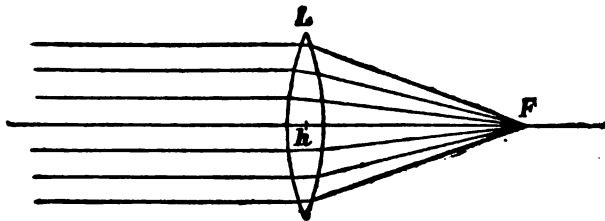
with their apices together at the centre of the lens (Fig. 7). From the fact that prisms deflect the rays towards their bases it will be seen that convex lenses will concentrate rays whereas concave lenses will disperse them.

The unit of measurement of dioptric lenses is a lens with a focal length of 1 metre ( $39\frac{3}{8}$  in.). This is termed a 1 dioptr (D.) lens. A lens having a focal length of 0.5 metre (20 inches) being twice as strong is termed a 2 dioptr lens, and one of 0.25 metres (10

inches) focal length a 4 dioptré, while a lens having a focal length of 2 metres, being only half the strength of a 1 D., is an 0.50 D., and one of a focal length of 4 metres an 0.25 D., etc.

The principal axis of a lens is a line passing through its centre perpendicular to its surface. A ray of light taking this direction is not refracted. Rays of light passing through a convex lens all converge to a point on its principal axis termed its principal focus.

FIG. 8.

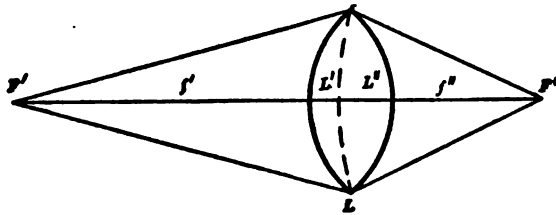


Union of parallel rays, refracted by a convex lens.

The distance of this point from the lens is termed the focal length of the lens (Fig. 8).

Divergent rays, that is rays coming from a distance less than infinity (6 metres; 20 feet) entering a convex lens are brought to a focus at a point farther from the lens than its principal focus. And if the rays emanate from a point twice the distance of the principal focus they will come to a focus an equal distance on

FIG. 9.



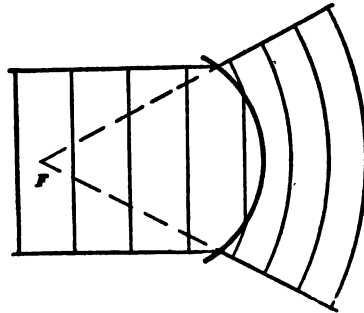
the other side of the lens. The nearer to the lens the point from which the rays emanate the farther from it on the opposite side will be the point to which they converge. These points are termed conjugate foci (Fig. 9).

Parallel rays passing through a concave lens are bent away from the principal axis and the principal focus must therefore be found by continuing backwards these divergent rays until they

meet, which will be, of course, on the side of the lens from which they emanated (Fig. 10).

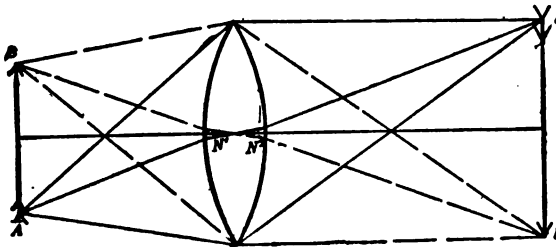
The image formed by a lens is the collection of the foci of the rays of light proceeding from the various points of the object. In a convex lens, if the object is distant the image will be formed

FIG. 10.



at the principal focus, and is termed a real image (Fig. 11). It will be on the side of the lens opposite the object so long as the object is at a greater distance than the focal length of the lens. If it is at a distance less than the focal length then the rays from the object will diverge after passing through the lens and no real image will be formed but a virtual image, erect and larger than

FIG. 11.



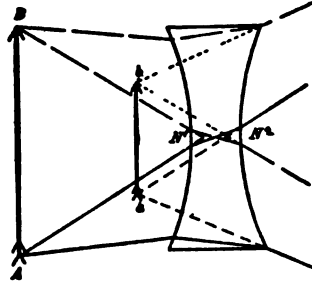
the object will be formed on the same side of the lens as the object. In a concave lens as the rays from an object are always divergent after passing through the lens the image is always virtual (Fig. 12).

*Refraction by the Eye.*—Although the dioptric system of the eye is composed of a series of convex lenses, for our purpose we may consider the three refracting surfaces—the surfaces of the

cornea, of the crystalline lens, and of the vitreous humor together as forming a convex lens having a focus of 23 mm. (0.92 inch) (Fig. 13).

As we have just learned the image formed by a convex lens is an inverted one so the images formed on the retina are inverted

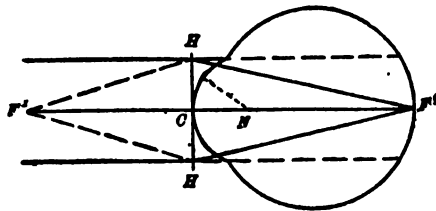
FIG. 12.



(Fig. 14). That they are interpreted by the brain as upright is probably the result of education.

Parallel rays of light, or rays of light coming from an infinite point, *i.e.*, situated at a distance of 6 M. (20 feet) or more, passing through the lenses of the eye with accommodation in abeyance, come together at the posterior or principal focus of the eye. If the posterior focus of the eye falls upon the retina, the eye is

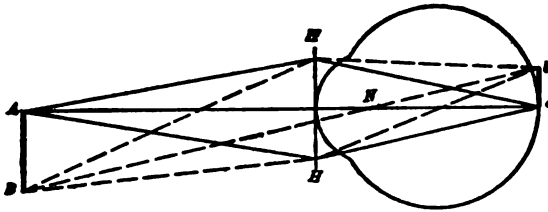
FIG. 13.



*emmetropic* (Fig. 15). If it falls behind it, the eye is *hyperopic* (Fig. 16). If it falls in front of it, the eye is *myopic* (Fig. 17). The last two conditions constitute *ametropia*. If the foci of no two meridians are the same, but each meridian focuses parallel rays at a different distance, the eye is *astigmatic*. In this condition all of the foci may be in front of the retina, or all behind it, or the focus of one meridian may be upon the retina and that of all of

the other meridians either behind or before it. If there were no way by which the focus of the eye could be altered, it would follow from what has been said that the only eye which could see distant objects distinctly would be the emmetropic eye. However the eye possesses in the ciliary muscle the power for altering its refractive state and thereby the position of the focus of the eye. This is

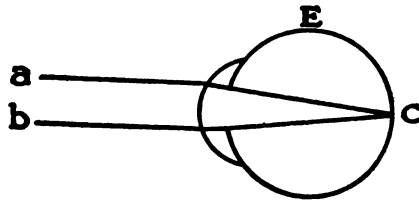
FIG. 14.



known as the power of accommodation. By the exercise of this function the crystalline lens is rendered more convex, thus increasing the refractive power of the eye (Fig. 18).

The power of accommodation is expressed in dioptries. The reason for this can be understood if we take for example an emmetropic eye without the power of accommodation. We have seen that such an eye under this condition can bring to a focus

FIG. 15.

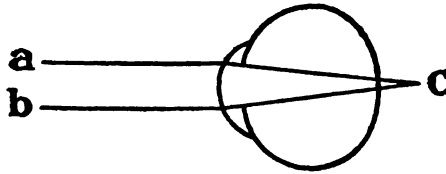


on the retina only parallel rays. We have also seen that if we place an object at the focus of a convex lens the rays coming from it emerge from the lens in a parallel direction. So that if we place before such an eye a lens whose focal length corresponds to the distance at which the object is from the eye the rays coming from it will emerge from the lens parallel and entering the eye will come to a focus on the retina, thus showing that had the refractive power of the crystalline lens, through the power of the accom-

modation been increased to an amount equal to the strength of the artificial lens, the object would have had its image formed upon the retina (Fig. 19).

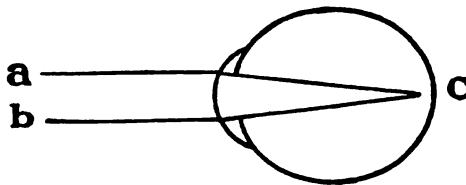
The power of accommodation is determined clinically by finding the nearest point at which the smallest type readable by the patient can be made out. The patient is told to bring the reading matter closer and closer until it blurs. The distance of this point

FIG. 16.



is then measured from the outer angle of the eye. This is known as the *punctum proximum* or near point of accommodation and represents the utmost contraction of the ciliary muscle. The *punctum remotum* represents the focus of the eye when it is adapted to its far point or when the ciliary muscle is completely relaxed. In emmetropia this is at infinity; in hyperopia behind the eye, and in myopia at some point between infinity and the eye. For an

FIG. 17.



emmetropic eye a lens of the focal length equal to the distance of the punctum proximum gives the power of accommodation. In hyperopia the power of accommodation is equal to a lens of the focal length of the punctum proximum plus the degree of the hyperopia. In myopia it is equal to a lens of the focal length of the punctum proximum less the amount of the myopia. In other words the accommodation  $(A) = P - R$ ,  $P$  corresponding to the punctum proximum and  $R$  to the punctum remotum. At first

sight it might be supposed that this formula did not apply to hyperopia as it was stated that the amount of the hyperopia was to be added to the near point. But it will be remembered that the punctum remotum ( $R$ ) in hyperopia is behind the eye and is therefore a negative or minus quantity so that the formula stands  $A = P - (-R)$  which  $= P + R$ .

The practical value of measuring the accommodation aside from the information it gives as to the actual power of the accommodation is the assistance it renders in determining the refraction of the eye. From a large number of determinations it has been found that from the tenth year of life there is a gradual constant loss of the accommodative power from loss of elasticity of the lens.

The power or range of accommodation for different age periods is given in the following table:

Age	Range of Accom. in Dioptres	Near Point	
		Centimetres	Inches
10	14.00	7.0	2.75
15	12.00	8.5	3.25
20	10.00	10.0	4.00
25	8.50	12.0	4.75
30	7.00	14.0	5.50
35	5.50	18.0	7.00
40	4.50	22.0	8.50
45	3.50	28.0	11.00
50	2.50	40.0	15.75
55	1.75	57.0	22.50
60	1.00	100.0	40.00
65	0.50	200.0	80.00
70	0.25	400.0	160.00

For the purpose of memorizing it may be pointed out that for the first three periods the difference between the power of accommodation is 2 D., for the next three it is 1.5 D. and for the next four 1 D.

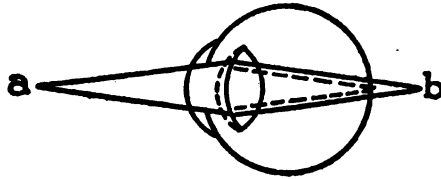
Suppose that a child 15 years of age is found to have a range of accommodation  $= 10$  D., a glance at the table shows that at this age it should be 12 D. Here is a deficiency of 2 D. which if not due to disease indicates the existence of  $H. = 2$  D. Suppose the same aged patient should be found to have a range  $= 14$  D. This shows an excess of 2 D. which would indicate a  $M. = 2$  D.

It will be noted in the table that at the age of 45 the range of accommodation  $= 3.5$  D. This represents a near point of 28



cm. (11 inches). Most near work is done at a distance of about 37 cm. (15 inches) or by the use of 2.5 D. accommodation, so that it is clear that the individual will have very little reserve accommodation while at work. As it is impossible for anyone to continuously exert his utmost muscular power but for a short time without experiencing fatigue it will be found that most

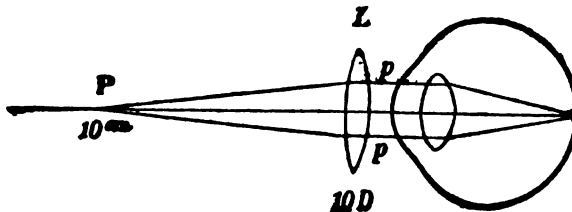
FIG. 18.



persons with emmetropic eyes or those rendered so by glasses will at this time of life require the addition of a convex lens to permit them to continue such work with comfort.

*Symptomatology.*—What are the symptoms indicating an error of refraction requiring correction? The principal ones are headache and asthenopia or eye-strain. There is nothing characteristic in the nature of the headache except that it is induced or aggravated

FIG. 19.



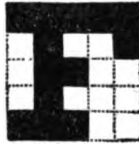
The accommodation replaced by a lens of glass, *L*, set before the eye.

by prolonged near work. It may be frontal, temporal, vertical, or occipital but is more commonly frontal, situated more particularly over the supra-orbital region. Morning headaches are not infrequently due to eye-strain resulting from the excessive use of the eyes the night before. Ocular pain or soreness of the eyeballs results from protracted near work. Fidgets is a very common symptom of eye-strain.

Muscular anomalies cause reflex symptoms which may be here

considered as these are disturbances often arising from errors of refraction. Headache, basal or extending from the eyes to the occiput; pain between the shoulder-blades; confused vision frequently called forth by car riding, sightseeing, and shopping, and often causing anxiety in the turmoil of crowded streets, conscious-

FIG. 20.

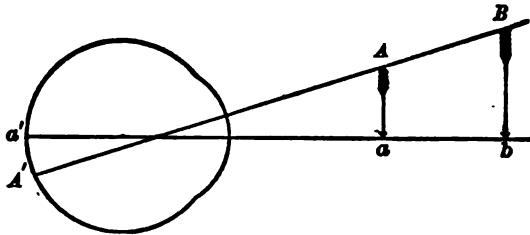


A letter from Snellen's test card.

ness on the part of the patient that the ocular muscles are not working in harmony, nausea and dizziness, and even vomiting if the strain is continued.

The headaches produced by inflammation of the sinuses are apt to be attributed to errors of refraction. By taking a careful history of the case it will often be possible to bring out symptoms

FIG. 21.



The visual angle.—The extremities of the object  $Aa$ , at the distance  $A$ , are projected on the retina at  $A'a'$ , while the extremities of the larger object  $Bb$ , at the distance  $B$ , by forming the same angle at the nodal point, are projected at points of the retina equally separated with those of  $Aa$ .

pointing to interference with breathing and disturbed secretions. It has been the writer's experience on at least three occasions to have patients come to him for glasses because of headaches of a week's duration in whom a listless manner and a history of nose bleed has aroused suspicion, and on taking the temperature the presence of fever was discovered which eventually proved to be typhoid.

Errors of refraction are seldom accompanied by failing vision except in presbyopia or old-age sight and when this symptom is complained of, a suspicion of some disturbance in the media or in the coats of the eye is to be suspected.

Periodic attacks of foggy vision in persons past 40 years of age should give rise to fears of glaucoma. In younger persons this phenomenon is often caused by a mild conjunctivitis in which a scant secretion floats over the surface of the cornea. The presence of glaucoma would be further suggested if the external examination should show a shallow anterior chamber, moderately large pupils, a dilatation of the vessels which come into view on raising the lids and exposing the sclera, by anæsthesia of the cornea, and by an increase in the tension of the globe which may be determined by palpating it through the closed lids by pressing alternately with the forefingers of both hands placed close together.

Gradual failure of vision in a person past 50 years of age is commonly due to incipient cataract. Here there may be also a shallow anterior chamber and the pupils may have lost their lustre.

Failing vision with flashes of light or phosphenes or a bluish flame before the eye is often complained of in retinochoroiditis.

Sudden loss of vision over part of the field with flashes of light, particularly if the fellow eye is myopic, should arouse suspicions of detachment of the retina. Most other intra-ocular disturbances are accompanied by external manifestations of inflammation.

*Systematic Examination of the Patient.*—It is the fault of many practitioners not to allow a patient to present his own story of his ills. Of course the latitude permitted patients must depend upon their intelligence, but to discount what the patient tells you because of preconceived ideas as to what should be complained of in a given disease is to interfere with progress. For instance in a typical case of interstitial keratitis one would be very apt to give little heed to the mother's statement that the inflammation followed an injury to the eye and yet we now know that in some cases this is the exciting cause in lighting up the trouble. Should the patient state that he "sees all right" but that upon reading for a time the print blurs and runs together; that after resting the reading can be resumed only to be followed in a short time

by a return of the confusion accompanied by pain in the eye, frontal distress and restlessness, we may suspect hyperopia. If he complains of not seeing well at a distance and of being obliged to hold reading matter too close to the eyes he is probably myopic. If he sees poorly both at a distance and near and has the troubles complained of by the hyperope he is probably astigmatic.

The second step in the examination should consist in a careful inspection of the eyes and the facial conformation. If the eyes appear small and deep-set they are probably hyperopic; if prominent, probably myopic. Marked facial asymmetry is indicative of astigmatism. If one eye is convergent high hyperopia usually exists; if divergent, myopia.

At this point it is well to use the "pin-hole test." We have in this test a valuable means of determining whether in an eye with poor vision this is due to a refractive error. We have learned that rays of light entering the eye along the principal axis undergo no refraction. Therefore if we exclude all but these rays, it will not matter what the error of refraction may be, if there be no disease of the eye vision will be approximately normal. So, blocking one eye, we place before the other eye a disk with a central pin-point opening, making sure that this is directly in the centre of the pupil, if the patient then is unable to read approximately the normal line on the test card it is almost a certainty that there is some other cause for the defective vision than an error of refraction.

*Testing of Visual Acuity.*—The next step in the routine examination is the testing of the visual acuity. The standard of normal vision is the ability to recognize letters which subtend an angle of 5 minutes, each stroke of the letter being made to subtend an angle of 1 minute (Fig. 20). This is based upon the fact that the eye cannot resolve or recognize, two dots unless they be separated by an interval of about 1 minute. Clinically the visual acuity is determined from cards containing rows of letters or characters of progressively smaller size from the top to the bottom of the card. Each row has designated above it the distance at which its letters subtend an angle of 5 minutes, or in other words the distance at which it should be seen by the normally refracting eye (Fig. 21).

The visual acuity is expressed by a fraction whose numerator represents the distance between the patient and the card, and whose denominator represents the distance at which the smallest row of letters read by the patient should be seen by the normally refracting or emmetropic eye. For example if the patient is placed 6 M. (20 feet) away from the card and is able to read the line which should be read at that distance the numerator becomes 6 (20) and the denominator 6 (20) the visual acuity being  $6/6$  or  $20/20$ . Supposing that at 6 M. (20 feet) the patient can see no lower than the line that should be read at 12 M. (40 feet), then the fraction becomes  $6/12$  or  $20/40$ . Suppose that at 6 M. (20 feet) the patient can distinguish no letters on the card. It will then be necessary for the patient to approach the card, and we note the distance from the card at which the patient first recognizes the largest letter on the card. Should the distance be 1.5 M. (5 feet) and the distance at which the letter should be read be 15 M. (50 feet) the visual acuity is expressed  $1.5/15$  or  $5/50$ . When the vision is reduced beyond the recognition of letters it is expressed in the distance at which fingers can be counted or if this is impossible it is expressed as light perception.

In testing the visual acuity a range of 6 M. (20 feet) should be used if available, but 5 M. (15 feet) or even 4 M. (12 feet) may be used without materially impairing the results. The card should, if possible, be illuminated by artificial light as this affords a constant intensity. If daylight must be employed the card should be placed upon a wall opposite a window. A standard of vision should be obtained for the quality of the light at the time of the examination by the examiner finding out what represents normal vision for himself under the given conditions. This applies also for artificial light. It will be found that by good artificial illumination the great majority of corrected eyes have a visual acuity in excess of what is accepted as the average normal. That which represents the usual vision obtained under the conditions employed should be determined and this considered as the normal.

The card should be so hung that the line representing normal vision for the distance employed is about on the line of the patient's eyes. If daylight is used the patient should be placed with his back

to the window. Each eye should be tested separately, the other eye being covered by a card resting upon the brow.

What will the visual acuity teach us concerning the state of the refraction? If the patient has normal vision the eye may be emmetropic or hyperopic. It can be neither myopic nor astigmatic except to a slight degree. If vision is sub-normal it may be myopic, astigmatic, or highly hyperopic, or there may be a spasm of accommodation. It is a good plan to have the patient start reading from the top of the card, as often it will be found that long before he has reached the line representing the limit of his visual acuity he has begun miscalling certain letters in each line, showing plainly that certain strokes are not seen distinctly. This is significant of the presence of astigmatism.

If normal vision may mean either emmetropia or hyperopia how can they be distinguished one from the other? Place before one eye, the other being closed, a 0.50 D. convex spherical lens. If the eye is emmetropic the vision will be made worse. If hyperopic vision will be unchanged or possibly improved. If the eye is found to be myopic vision will be improved by concave lenses. If the eye is astigmatic vision will be but little improved by either convex or concave spherical lenses.

Having recorded the vision and obtained what information we can from it we proceed to measure the power or range of accommodation. There are several methods recommended for the purpose. The most practical one, though perhaps not the most accurate, is by the use of reading test-type, constructed on the principle of the distance type, but arranged in paragraphs and of such size that the normal vision is represented by the ability to read the different paragraphs at distances varying from 0.25 M. (10 inches) to 2 M. (80 inches). Each eye is tested separately, the smallest type which the patient can read being used. The nearest point and the farthest point at which this type can be read is then determined and measured from the outer angle of the eye. The test is fairly accurate in determining the amount of hyperopia or the amount of myopia present.

It may be recalled that in myopia the rays coming from an object at the far point of the eye come to a focus on the retina, and that a lens of the focal length of the distance of this point

from the eye will give to parallel rays a direction as though they came from this point; therefore a lens of this focal length will correct the myopia. Suppose that the farthest point at which the patient can read is 25 cm. (10 inches), then a lens having this focal length, a 4 D. lens, will correct the myopia.

In hyperopia the nearest point at which the patient can read will represent the accommodative power of the eye. It has been shown that the accommodative power bears a pretty constant relation to the age of the patient so that the difference between the accommodation that the patient has and that which he should possess at his age represents the amount of accommodation expended in the correction of his hyperopia. Suppose that the patient is 20 years of age. At this age the accommodation is 10 D. Suppose the nearest point to which he can bring the print is 16.5 (6½ inches). This represents an accommodation of 6 D. Then the difference between 10 D. and 6 D. represents the amount of hyperopia or 4 D.

In astigmatism the patient will be unable to read the fine print or will hold it very close in order to obtain large retinal images.

# State Medicine

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## THE LAW RESPECTING COMPENSATION TO WORKMEN FOR ACCIDENTS IN GREAT BRITAIN, AND ITS OPERATION

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A WORKMAN in the United Kingdom who receives an injury caused by accident arising out of and in the course of his employment, which prevents him from following his employment, has redress against his employer either (a) at common law, (b) under the Employers' Liability Act, 1880, or (c) under the Workmen's Compensation Act, 1906, which repealed the Acts of 1897 and 1900 of similar title.

The liability of employers to workmen for injuries resulting from accident is determined under the Compensation Act by certain carefully defined provisions. The general principle of that Act is this: If in the course of employment and arising out of the employment personal injury is sustained by a workman, his employer shall be liable to pay compensation in accordance with certain other carefully defined provisions, subject to the following exceptions: (a) The employer shall not be liable for compensation respecting an injury which does not disable the workman for at least one week from earning full wages at the work at which he was employed; (b) nor where it is proved that the injury is attributable to the serious and wilful misconduct of the workman, except where the injury results in death or serious and permanent disablement. When the injury is caused by the personal negligence or wilful act of the employer or of some person for whose act or default the employer is responsible, the civil liability to compensation of the employer remains, but in that case the workman may, at his option, either claim compensation under the Compensation Act, 1906, or



he may take proceedings independently of that Act, that is, either at common law, or under the Employers' Liability Act.

It may happen that while the employer is liable in civil law for a fatal accident to a workman which has been occasioned by the act or omission of a fellow-workman, the defaulting workman may be charged under the criminal law with manslaughter in England or culpable homicide in Scotland. In such fatal accidents, an inquiry is made in England by the Coroner, and in Scotland, under the Fatal Accidents Inquiry (Scotland) Acts, 1895 and 1906, by a sheriff and a jury.

So far as civil liability, however, is concerned, that is determined and delimited by the decision of the civil court respecting the action for compensation as raised by the workman, or by his relatives in the case of his death, under any one of the ways already named. Should a workman, or his relatives, as the case may be, raise an action independently of the Compensation Act to recover compensation for the injuries sustained, and should it be determined by the Court that the employer is not liable in such action but that he would have been liable under the Compensation Act, the Court shall dismiss that action as raised, but should the plaintiff so choose, the Court shall proceed to assess such compensation as if the action had been raised under the Compensation Act, subject, however, to deduction therefrom of all or part of the costs incurred in bringing the action instead of proceeding under the Compensation Act, and shall certify as to the compensation awarded and the direction given as to deduction of costs, and such certificate shall have the force and effect of an award under the Compensation Act. Such an arrangement prevents a miscarriage of the law relative to the workman, secures to him his legal compensation, and protects the employer against unnecessary litigation.

The Compensation Act further determines the time within which proceedings shall be taken by a workman who has received an injury from accident during his employment. It enacts that proceedings for recovery of compensation shall not be maintainable unless "notice of the accident has been given as soon as practicable after the happening thereof and before the workman has voluntarily left the employment in which he was injured, and unless the claim for compensation with respect to such accident has been made within

six months from the occurrence of the accident causing the injury, or, in the case of death, within six months from the time of death." But it is also declared that the want of such notice or any defect or inaccuracy in it shall not be a bar to proceedings if it is found that, on a notice or an amended notice being then given and the hearing of the case postponed, the employer would not be prejudiced in his defence, or that the want, defect, or inaccuracy of such notice was occasioned by mistake, absence from the United Kingdom, or other reasonable cause; and the failure to make a claim within the six months shall not be held to be a bar to proceedings if such failure was due to any of the aforementioned reasons.

The notice shall give the name and address of the workman, shall state in ordinary language the cause of the injury, and the date at which the accident happened, and shall be served by delivering the same at, or by sending it by post in a registered letter addressed to, the residence or place of business of the employer.

The Compensation Act makes provision for "contracting-out"; but this becomes operative only under a certificate of the Registrar of Friendly Societies that any scheme of compensation, benefit, or insurance is financially suitable, and that its substitution is of mutual consent of employer and workmen. Such a scheme must, where it provides for the contributions of employees, confer benefits at least equivalent to such contributions over and above the benefits to which the employees would be entitled under the Act.

Where an employer acting as principal contracts with any contractor for the execution of all or any work undertaken by the contractor, the principal shall be liable to pay compensation to any workmen of the contractor injured in such work, the amount to be fixed by the earnings of the workmen under the contractor; but when the contract relates to threshing, ploughing, or other agricultural work and the contractor provides and uses machinery driven by mechanical power for such work, the contractor alone shall be liable to pay compensation.

The Act also contains provisions to ensure payment of compensation in the case of companies or employers whose affairs are in liquidation or bankruptcy, as well as whereby a workman may seek remedy both against his employer and some other person, where there are circumstances creating a liability in that other person.

The benefits of the Compensation Act apply to all persons who are in employment, including servants in domestic service except those whose employment is merely casual, and including all persons employed in the private service of the Crown or in any workshop under the control of the Government. They also apply to masters, seamen, and apprentices to the sea service and to apprentices in the sea-fishing service of all vessels registered in the United Kingdom or of any other British ship or vessel of which the owner or manager resides or carries on business in the United Kingdom, and to pilots in terms of Part X of the Merchant Shipping Act, 1894. But they do not apply to members of the crew of a fishing vessel who are remunerated by shares in the profits or gross earnings of the working of such vessel, nor do they apply to persons in the naval or military service of the Crown. Lest differences may arise regarding the interpretation of the expression "workman," the term has been defined in the Act as follows: "*Workman* does not include any person employed otherwise than by way of manual labour whose remuneration exceeds two hundred and fifty pounds a year (1250 dollars) or a person whose employment is of a casual nature and who is employed otherwise than for the purposes of the employer's trade or business, or a member of a police force, or an out-worker, or a member of the employer's family dwelling in his house, but, save as aforesaid, means any person who has entered into or works under a contract of service or apprenticeship with an employer, whether by way of manual labour, clerical work, or otherwise, and whether the contract is expressed or implied, is oral or in writing." It is thus apparent that the benefits of the Act practically extend to the whole army of labor.

But in addition to these provisions which have solely to do with injury after accident in the ordinary sense, the Act further makes provision for compensation to workmen who are disabled by or are suspended from their usual employment on account of having contracted a disease named in the Third Schedule of the Act or a disease which may have been added to that list at some time by the Home Secretary subsequent to the passing of the Act in virtue of powers conferred upon him by the Act.

The diseases to which the Act at the present time applies are as follows.

## WORKMEN'S COMPENSATION ACT, 1906

## THIRD SCHEDULE

DESCRIPTION OF DISEASE	DESCRIPTION OF PROCESS
Anthrax:	Handling of wool, bristles, hides, and skins.
Lead poisoning or its sequelæ:	Any process involving the use of lead or its preparations or compounds.
Mercury poisoning or its sequelæ:	Any process involving use of mercury or its preparations or compounds.
Phosphorus poisoning or its sequelæ:	Any process involving the use of phosphorus or its preparations or compounds.
Arsenic poisoning or its sequelæ:	Any process involving use of arsenic or its preparations or compounds.
Ankylostomiasis:	Mining.

## STATUTORY ORDER OF HOME SECRETARY

MAY 22, 1907. No. 407

DESCRIPTION OF DISEASE	DESCRIPTION OF PROCESS
1. Poisoning by nitro- and amido-derivatives of benzene (dinitrobenzol, anilin, and others) or its sequelæ:	Any process involving the use of any such derivatives of benzene, or its preparations or compounds.
2. Poisoning by carbon bisulphide or its sequelæ:	Any process involving the use of carbon bisulphide or its preparations or compounds.
3. Poisoning by nitrous fumes or its sequelæ:	Any process in which nitrous fumes are evolved.
4. Poisoning by nickel carbonyl or its sequelæ:	Any process in which nickel carbonyl gas is evolved.
5. Arsenic poisoning or its sequelæ:	Handling of arsenic or its preparations or compounds.
6. Lead poisoning or its sequelæ:	Handling of lead or its preparations or compounds.
7. Poisoning by African boxwood ( <i>Gonioma kamassi</i> ) or its sequelæ:	Any process in the manufacture of articles from that wood.
8. Chrome ulceration or its sequelæ:	Any process involving the use of chromic acid or bichromate of ammonium, potassium, or sodium, or their preparations.

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| 9. Eczematous ulceration of the skin produced by dust or caustic or corrosive liquids, or ulceration of the mucous membrane of the nose or mouth produced by dust: |   |
| 10. Epitheliomatous cancer or ulceration of the skin or of the corneal surface of the eye, due to pitch, tar, or tarry compounds:                                  | Handling or use of pitch, tar, or tarry compounds.                                      |
| 11. Scrotal epithelioma (chimney-sweeps' cancer):  | Chimney-sweeping.   |
| 12. Nystagmus:   | Mining.   |
| 13. Glanders:  | Care of any equine animal suffering from glanders; handling the carcass of such animal. |
| 14. Compressed-air illness (caisson-disease) or its sequelæ:   | Any process carried on in compressed air.   |
| 15. Subcutaneous cellulitis of the hand (beat hand):   | Mining.   |
| 16. Subcutaneous cellulitis over the patella (miners' beat knee):  | Mining.   |
| 17. Acute bursitis over the elbow (miner's beat elbow):  | Mining.   |
| 18. Inflammation of the synovial lining of the wrist-joint and tendon sheaths:   | Mining.   |

#### STATUTORY ORDER OF HOME SECRETARY

##### DESCRIPTION OF DISEASE

##### DESCRIPTION OF PROCESS

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| 1. Cataract in glass-workers:  | Processes in the manufacture of glass involving exposure to the glare of molten glass. |
| 2. Telegraphists' cramp:   | Use of telegraphic instruments.  |
| 3. Eczematous ulceration of the skin produced by dust or liquids or ulceration of the mucous membrane of the nose or mouth produced by dust. | Substituted for No. 9 of Order of May 22, 1907.  |

According to the terms of the Factory and Workshop Act of 1901, any of the first five industrial diseases named in the Third Schedule as hereinbefore stated when they occur must be reported by any medical practitioner to the Chief Inspector of Factories, for which a small fee is paid to the practitioner. According to Section 8 of the Compensation Act, where a certifying surgeon appointed under the Factory Act certifies that a workman is suffering

from, or has been suspended from his employment because he has contracted, or has died from, any of the aforementioned industrial diseases, such workman or his relatives shall be entitled to compensation as if the disease were a personal injury by accident arising out of and in the course of his employment, provided that at the time of entering such employment such workman has not wilfully and falsely represented in writing that he has not previously suffered from such disease. The compensation in such cases is recoverable from the employer who last employed the workman during twelve months within which the disablement, suspension, or death occurred, but should any employer allege that the workman's disease was in fact contracted whilst the workman was in the employment of some other employer and not whilst in his employment, that other employer may be joined with him in the arbitration, and the amount of the liability of each to the workman may be determined by arbitration under the Act. If a workman at or immediately after his disablement or suspension was employed in any process named in the second column of the foregoing schedules, and the disease causing his disablement or suspension is the disease named in the first column opposite the description of the process, the disease shall be deemed to have been due to the nature of that process, except where the certifying surgeon certifies that in his opinion the disease was not so due, or except the employer proves to the contrary.

**MEDICAL REFEREES.**—In view of the fact that in order to establish a claim for compensation medical evidence must of necessity be called for, and the further fact that the Courts must have some conclusive evidence of a medical kind where medical witnesses differ in opinion, the Act empowers the Home Secretary to appoint for the purposes of the Act such legally qualified practitioners as he may determine to be medical referees, whose remuneration and expenses incurred under the Act are to be paid out of moneys provided by Parliament. It has been laid down as a condition of appointment by the Home Secretary that the persons to be appointed shall not retain any appointment which would be likely to interfere with their duties as referee or to bias their judgment. Where a medical referee has been employed as a medical practitioner in any case by or on behalf of an employer or a workman or by any insurers interested, he cannot act as medical referee in that case.

According to the terms of Paragraph 15 of the First Schedule of the Act, the certificate of a medical referee "shall be conclusive evidence as to the matters so certified." The Courts have ruled accordingly. In the case of *Ferrier v. Gourlay Brothers* [4 F. (5th series), 711], the Court of Session (Scotland) held that the report of the referee was conclusive evidence of the workman's condition at the date of the report; and in the Court of Appeal, London, Nov. 26, 1907, in the appeal of *Bromilow and Co.*, against a decision of the County Court judge on this point, the Court held that the lower court was in error in holding that the certificate of the referee was not conclusive evidence of the condition of the workman. Further, in respect that in not a few cases which come before the Courts evidence sometimes of a highly technical medical character requires to be given, Paragraph 5 of the Second Schedule of the Act provides that a judge of County Courts in England or Ireland, or the Sheriff or his Substitute in Scotland, may, if he thinks fit, summon a medical referee to sit with him as an assessor. This arrangement is calculated to be of considerable advantage not only to the judge but also to both parties in the case in the administration of justice.

**METHOD OF ARBITRATION.**—For the purpose of settling any question of compensation arising under the Act, if a committee representative of employer and workman exists with powers to settle matters under the Act, such committee shall arbitrate, provided that no written objection is sent by either party before the committee meet for the purpose of settling. Should no such objection be taken, and should the committee fail to effect a settlement, or should there be no such committee, the matter shall be settled by a single arbitrator agreed on by the parties, or in the absence of agreement to appoint such an arbitrator, by the judge of a County Court in England or Ireland and by a Sheriff or Sheriff-substitute in Scotland, in accordance with the procedure laid down in the Act. While such provisions as the foregoing with respect to an arbitration committee or the appointment of a single arbitrator are found in the Act, in practice such cases when contested are usually brought before a judge or a sheriff.

**AMOUNT OF COMPENSATION TO BE PAID.**—This has been carefully defined and delimited in the Act. Differentiation is naturally made as between the amount to be paid (*a*) where the acciden-

tal injury is fatal, and that (b) where the injury produces disablement or incapacity of a temporary or of a permanent character. Where death results from the injury, and where the deceased workman leaves anyone wholly dependent upon his earnings, the amount to be paid shall be a sum of money equal to his earnings in the same employment of the same employer for the three years preceding the date of his injury, or the sum of £150 (750 dollars), whichever sum is the larger, but not exceeding in any case £300 (1500 dollars), provided that any moneys paid to the workman after his injury and prior to his death shall be deducted therefrom. If the period of employment of the workman by the same employer be less than three years, then the sum shall be one hundred and fifty-six times the average weekly earnings during the period of actual employment.

Where the workman leaves only others partly dependent upon his earnings, the payment shall be such sum as before, in the case of agreement, or such sum as shall be determined to be reasonable and proportionate to the injury of such dependents by arbitration under the Act.

Where a workman leaves no dependents, the payment in case of death shall be made to his legal personal representative, or where there is no such representative, to the person to whom the expenses of medical attendance and burial are due.

Where total or partial disablement for work results from the injury, the sum to be paid shall be a weekly payment not exceeding fifty per cent. of the workman's average weekly earnings, but not to exceed one pound (five dollars); where the workman at the date of the injury is under 21 years of age and his weekly earnings are less than twenty shillings (five dollars), the amount to be paid shall not exceed half that amount.

The weekly payment may be reviewed by the Court from time to time at the request of either employer or workman, and on such review may be ended, diminished, or increased, subject in the latter case, however, to the maximum already stated, and in the case of a workman under 21 years whose disablement exists for more than twelve months, the Court on review may increase the amount of weekly payment to an amount not exceeding fifty per cent. of the weekly earnings he would probably have been earning but for the accident.



Where the disablement is partial, so that the workman is able to follow some other but less remunerative occupation than his normal occupation, the weekly payment shall in no case exceed the difference between the average weekly earnings of his interim and normal occupation, but shall bear such relation to the amount of that difference as is proper under the circumstances.

If a workman has been in receipt of weekly payment for not less than six months, his employer may apply to the Court to rule that the liability therefor be discharged or redeemed by the payment of a lump sum of such an amount as, where the incapacity is permanent, would, if invested in the Post Office Savings Bank, purchase an annuity equal to seventy-five per cent. of the annual value of the weekly payment.

If a workman leave the United Kingdom while in receipt of a weekly payment, such payment shall cease, unless it has been certified by a medical referee that the incapacity is likely to be of a permanent nature.

WHAT IS AN ACCIDENT?—While the Compensation Act speaks of personal injury *by accident*," "injury to a workman *by accident* arising out of and in the course of the employment," and "injury caused by *any accident*," it is absolutely silent in respect of any definition of what is an accident. Neither does French law define it; but in French official circulars the following may be observed: "Accident in our sense consists in a bodily injury arising from the sudden occurrence of an external cause." The law of Germany defines it thus: "An accident is an abnormal and strange happening in the course of the employment which occurs suddenly, and the consequences of which are injurious to life and health." In Great Britain, therefore, it has been left for the judges in the courts to determine in any given case whether an accident has taken place, and whether the accidental injury arose out of and in the course of the employment. It was perhaps not unwise that the Legislature should have left the term undefined, since an "accident" may differ greatly in its essence under different circumstances. What a judge has to determine in any case is, whether in the employment *personal injury by accident arose out of and in the course of the employment to the workman*, in order that compensation shall be paid. Under the Employers' Liability Act, an em-

ployer could plead that the workman at the time he received his injury was guilty of contributory negligence, but under the Compensation Act that is somewhat modified to the extent that "if it is proved that the injury is attributable to the serious and wilful misconduct of the workman, any compensation claimed in respect of that injury shall, unless the injury results in death or serious and permanent disablement, be disallowed." With this exception, if a judge finds that the accidental injury arose out of and in the course of the employment, compensation would legally follow. The chief bone of contention in many cases is not whether there has been personal injury, but whether the injury did so arise out of and in the course of the employment. Should one or other of these conditions not be satisfied, compensation would be denied. In respect of the absence of definition of the term "accident" in the Act, differences of opinion have arisen even among judges of the lower courts as to what, under certain sets of circumstances, constituted an accident, and, in consequence, such judgments have been appealed to the highest court, the House of Lords. In one such disputed case, Lord McLaren, one of the judges of the Court of Session (Scotland) gave the following view of an accident: "If a workman in the reasonable performance of his duties sustains a physiological injury as the result of the work he is engaged in, this is an accidental injury in the sense of the statute." (*Stewart v. Wilsons and Clyde Coal Co.*, 5F., 120.) In another case in which a workman in a weak state of health was permitted to work his way to England in the stokehold of an Atlantic steamer, wherein he succumbed to heat-stroke while acting as a stoker, and where the judgment of the lower Court was appealed to the House of Lords, the Lord Chancellor in giving judgment, said: "To my mind the weakness of the deceased which predisposed him to this form of attack is immaterial. The fact that a man who had died from a heat-stroke was by physical debility more likely than others so to suffer can have nothing to do with the question, whether what befel him is to be regarded as an accident or not. . . . "It was an unlooked-for mishap in course of his employment; in common language, it was a case of accidental death." The judgment in this case has ruled several subsequent cases; for example, in the case of *Hughes v. Glover, Clayton & Co.*, the County Court judge held, and the Court of Appeal upheld the decision, that

death was due to an accident. The circumstances were these: The workman was engaged in tightening a nut with a spanner when his foot was seen to slip and he himself to fall on his back. He was found to be dead. Post-mortem examination showed that death was due to rupture of a very large aneurism of the aorta. Medical evidence was to the effect that death might have happened at any time from very slight exertion, or, indeed, without any unusual exertion, as in a natural act.

But the term "accident" is legally capable of extension beyond its natural meaning. It has been ruled in the Irish Courts, in *Anderson v. Balfour*, that a gamekeeper who was wounded by poachers was entitled to compensation because his injury arose out of and in the course of his employment, and by the English Courts, in *Challis v. The London and South-Western Railway Co.*, that an engine-driver injured by a stone dropped by a boy from a railway bridge came likewise under the Act. Within the last month (July), also, the English Court of Appeal decided that the widow of the cashier of a colliery company was entitled to compensation for the death of her husband under the following circumstances. Her husband in the course of his duty was carrying in a railway train to a coal-pit some distance from the office of the company a large sum of money wherewith to pay the wages of the men. In the course of that journey he was murdered. It was held that his death was due to accident arising out of and in the course of his employment, notwithstanding that the act whereby his death was caused was a criminal and premeditated act. From what has been said, therefore, it is clear that to constitute an injury to be accidental, that injury must have arisen out of and in the course of the employment. Many cases might be cited in which it has been held that an accident has arisen in the course of the employment but did not arise out of the employment, but two examples will suffice. A miner was killed by an explosion of gas in a coal-pit. In accordance with the provisions of the Coal Mines Regulation Acts, the pit had been inspected that morning of the occurrence by the official pit-fireman. That official found gas in one part of the pit, and the deceased was informed of the fact. In addition, the fireman placed a barrier against admittance to that part, and on the barrier wrote in chalk the words "No road up here." The deceased disregarded

these notices, entered the place with a naked light, and so caused the explosion by which he was killed. The Judge held, notwithstanding that the plaintiffs founded their claim for compensation on the provision in the Compensation Act that serious and wilful misconduct is not available as a defence where death results from accident, that an accident arising in a place where the deceased was expressly forbidden to enter could not be said to arise out of his employment, and compensation, therefore, was refused. In the other case, which also happened in a coal-pit, the workman desiring to go to the surface, entered an up-going cage at the bottom without permission of the official in charge, and in the course of the ascent was injured. In the Court of Session, the Lord-Justice Clerk ruled that "the accident arose in the course of the employment, but did not arise out of it. It arose out of the wilful act of the respondent in breaking certain rules which he was 'bound to obey.'"

In addition to the foregoing class of cases, in a very large number of others litigation has ensued owing to the contention of employers either that the alleged injury was not sustained, or that the injury sustained was not of the gravity alleged.

Having thus reviewed the general tenor of the provisions of the Compensation Act, it will, perhaps, be of advantage to consider the machinery provided in the Act whereby compensation may be secured by an injured workman. The first necessary condition is the giving of notice of the accident and of his injury under the conditions already mentioned. Thereafter, the workman is examined by a medical practitioner of his own choice, or, if required by his employer, he shall submit himself for examination by a practitioner provided and paid by the employer. If the workman refuse to submit himself to that examination by the employer's practitioner, or in any way obstructs the same, his right to compensation or to take proceedings in relation thereto, shall be suspended until such examination has taken place. When a workman after receipt of his injury has submitted himself for examination by a medical practitioner either of his own choosing or by a practitioner selected by the employer, and when the employer or the workman, as the case may be, has within six days after such examination furnished the other with a copy of the report of such practitioner as to the workman's condition, then, in the event of no agreement being come

to between the employer and the workman as to the workman's condition or fitness for employment, the registrar of a county court in England or Ireland or the sheriff-clerk in Scotland, on application being made to the Court by *both* parties, may, on payment by the applicants of a prescribed fee not exceeding one pound (five dollars), refer the matter to a medical referee. The medical referee, after examination of the workman, shall give a certificate as to the condition of the workman and his fitness for employment, specifying where necessary the kind of employment for which he is fit (if any), and that certificate shall be conclusive evidence as to the matters so certified. Where no agreement can be come to between the employer and the workman as to whether or to what extent the incapacity of the workman is due to the accident, the reference to a medical referee shall apply as if the question were a question as to the condition of the workman; and if the workman, on being required so to do, refuses to submit himself for examination by the medical referee as aforesaid, or in any way obstructs the same, his right to compensation and to take proceedings shall be suspended until such examination shall have taken place. It will be observed that if the workman do not become a party to the application for a reference to a medical referee, he cannot be compelled so to become; but his right to compensation and to take or prosecute proceedings is thereby suspended unless and until he submits himself to examination by the referee. The practical effect, therefore, is to compel the workman to submit himself for examination. When a reference to a medical referee is made by the Court, the referee shall determine (1) as to the condition of the workman relative to his fitness for employment, (2) the kind of employment (if any) for which he is fitted, and (3) to what extent the incapacity was due to the accident. Besides, a medical referee may be called upon to decide whether said incapacity is or is not likely to be of a permanent character, so as to enable the Court to determine the amount of compensation to be paid.

Where a workman's claim for compensation has been ceded by the employer, and he is receiving weekly payments, he shall, if required by the employer, submit himself from time to time for examination by a medical practitioner chosen by the employer and paid by the employer, and if the workman refuse or obstruct such

examination, his right to the weekly payments shall be suspended until such examination has taken place.

The extension by the Compensation Act (1906) of the benefits of compensation to practically all classes of employees, including the domestic servant class, has compelled an enormous expenditure of money by employers of all classes in insurance against accidents. It is, perhaps, not possible to arrive at anything like an approximate estimate of the very large sums of money which have to be expended annually in this protection, but it may safely be set down as many millions of pounds. In addition to this, there is the further enormous outlay involved in defending and prosecuting claims for compensation; indeed, the Act has provided an enlarged and lucrative source of income to the legal profession, in which to some extent the medical profession has shared. The whole Act would seem, both financially and otherwise, to operate more favorably to the employee than to the employer. If a fraudulent or exorbitant claim, for example, is put forward, the burden of disproving the claim falls upon the employer. This is not seldom difficult, not only because of the circumstances in which the alleged injury has taken place, but also because of the nature of the injury alleged to have been sustained. This is especially true respecting injuries of the nature of sprains and strains to muscles and ligaments, of hernia, and other conditions, not to speak of the differences of opinion which may arise respecting the time of ability to resume employment after such injuries as fractures and dislocations of limbs.

The decision of the House of Lords that any personal defect or disease in a workman at the time of injury or death—defect or disease, which may indeed be directly contributory to, or actually invite, the injury or death—is no bar to compensation, opens up a very large question for employers. Experience has demonstrated that the age and fitness of employees exercise a definite influence in the relative production of injuries, and insurance companies and employers alike are gradually coming to the view that both of these factors in the usefulness of workmen must be more carefully scrutinized in the future than they have hitherto been. Some insurance companies, indeed, are now hesitating or declining to insure against accident workmen over the age of 45 years, because they have found that accidental injuries are more common over than

under that age. Should this policy gradually prevail, employers will be compelled to limit the age at which workmen shall be engaged or shall be permitted to continue at work. Initial personal defects, such as abnormal or defective vision, defective hearing, lameness or other limited physical defect, etc., will also act as limitations to employment, since workmen who suffer from these are more liable to accidents than normal workmen. Indeed, it would appear as if the time was soon to arrive when employers shall be compelled to insist upon a medical examination of workmen whom they engage, and to demand such examination at periodic intervals thereafter.

## PHYSICIANS' FEES DOWN THE AGES \*

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ANYONE who would persuade himself that it is only in our time that physicians have claimed and have obtained good professional fees, that is a reward for services rendered, dependent not on the amount of work done but on the amount of benefit conferred upon the person for whom the work is done, is sadly mistaken. From the very beginning of the history of medicine we find definite records of the professional character of the rewards accorded to physicians and a thorough recognition of the fact that those rewards depended not at all on the amount of labor required for what was accomplished, or even on the quality of the skill of the practitioner of medicine, but on the benefit that the patient felt that in his circumstances he had derived from the ministrations of this member of a learned profession.

The very first physician of whom we get a sufficiently accurate picture to enable us to have some idea of the success of his life-work shows us this very clearly. His name as it has come down to us was I-Em-Hetep. He was the physician to King Tchser, of the Third Dynasty in Egypt, and probably lived not later than 4500 B. C. He seems to have had admirable success in the practice of his profession. The names by which he was known included besides that already mentioned those of "master of secrets," "writer of mysteries," and then especially "bringer of peace." He seems to have been a psychotherapist in his power to soothe the mind. Besides the reverence of men for him, while he was alive, their memory of him remained after death, and he was placed by them among the gods. He was not deified however as the kings were, but retained more of his human characteristics.

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\* A lecture on the history of medicine at Fordham University School of Medicine.



His statues are to be found not bearded as those of the kings, but without a beard showing that it was the essential humanity of the man that his contemporaries had come to regard with particular reverence. For several thousand years he was worshipped in Egypt as one who had conferred great favors on mankind and who still might be able even from beyond the grave to extend a helping hand to those who were in affliction. The place that he secured in popular estimation shows how highly his professional services were valued.

After him the next great member of the medical profession of whom there is particular mention in history was a distinguished surgeon. He was buried in the old cemetery at Sakkara near Memphis and his tomb was in the so-called Step Pyramid. We know him as a surgeon from the fact that the walls of his tomb were decorated with pictures<sup>1</sup> of surgical operations. These are the first surgical operations of which we have any records and the first pictures of surgical operations extant. Among them is the opening of a carbuncle on the back of the neck showing that men suffered from the same diseases nearly 5000 years ago as they do now. There are also operations upon the hands and feet. The most prominent feature of the surgery is the evident effort on the part of the patient to suppress manifestations of pain. One hand is placed in the axilla of the arm on the opposite side of the body while the other hand is being operated upon. This surgeon seems to have been one of the high dignitaries of Egypt of that time, for it was only the high court officials who could secure the erection of a pyramid in their honor. The date of this pyramid to the first great surgeon of history is not later than 2500 B. C.

We know nothing at all about the amounts of money or other rewards that these old-time members of the profession received. All we know is that their rewards must have been ample since the recipients came to occupy such prominent places in the country at that time. The next important source of information that we have, however, though it comes from very old times, gives some very accurate details with regard to the fees paid to physicians

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<sup>1</sup>Those who are interested will find copies of these pictures in my article on them in the Jour. Amer. Med. Assoc., November 9, 1907.

and surgeons. This is to be found in the Code of Hammurabi who was the sixth King of the First Dynasty at Babylon and reigned for over fifty years about 2250 B. C. He is identified by most Assyriologists with King Aramaphel who is mentioned in Genesis xiv, 1. He was a great soldier according to the historians of his time and a wonderfully good ruler who having destroyed all his enemies of the north and the south enabled his people to dwell in peace. Prof. Robert Francis Harper, in his introduction to his translation of the Code of Hammurabi,<sup>2</sup> says of Hammurabi:

"He codified the existing laws that the strong might not oppress the weak, that they should give justice to the orphan and widow, and for the righting of wrong. He rebuilt cities and canals, he restored temples and endowed them with means for sacrifices, he reestablished cults, he reunited his people."

His Code of Laws was found on the Acropolis of Susa in 1902 by an expedition sent out by the French government. It is engraved upon a block of stone nearly eight feet high which was broken into three pieces but these were easily joined together. It is a revelation of the wonderful legislative ability of this old-time King. There is scarcely an important social problem of the modern time from divorce and the servant problem to the fees of professional men and the relations of relatives to one another in money matters, and the indemnities to be paid for injuries, and the settlement of damage cases, with regard to which Hammurabi did not draw up some very common-sense legislation. His Code was a triumphant expression of the justice of mind and the thoroughly legal temperament of the men of that old time. For us the question of the physicians' fees is the important element of the Code. The old King recognized particularly the necessity for valuing a physician's services professionally, that is according to the advantage which accrued to the patient from them. Accordingly, he does not proclaim that a particular operation is to be done for so much, but a particular operation on a wealthy person was one price, on a middle class person another price, on a working man a third price. Here are the exact words of the Code.

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<sup>2</sup> Robert Francis Harper: Chicago University Press, 1904.

"If a physician operate on a man for a severe wound (or make a severe wound upon a man) with a bronze lancet and save the man's life, or if he open an abscess (in the eye) of a man with a bronze lancet and save that man's eye, he shall receive ten shekels of silver (as his fee).

"If he be a freeman, he shall receive five shekels.

"If it be a man's slave, the owner of the slave shall give two shekels of silver to the physician."

For less important services rendered smaller fees were enacted. There is not much detail in the matter and there was evidently a simplicity about diagnosis in those early days, that must have made the application of so simple a code easy. Probably all the internal troubles were placed together under a few categories. The most important of these in Egypt would surely be, as it is at the present time, affections of the bowels. This is the only internal medical trouble that is mentioned then, but a rather good fee is provided for it.

"If a physician set a broken bone for a man or cure his diseased bowels, the patient shall give five shekels of silver to the physician.

"If he be a freeman, he shall give three shekels of silver.

"If it be a man's slave, the owner of the slave shall give two shekels of silver to the physician."

The value of these fees can of course only be estimated by the buying power of money at that time. The shekel is a coin which was adopted from the Assyrians by the Hebrews and the Phœnicians, but varied much in different countries. Originally the term was used as a unit of weight by the Babylonians and was equal to about 250 grains troy. Ten shekels of silver then would probably not be worth much more than five dollars and this does not seem a very suitable reward for saving a man's life or his eye. The real value of it however must be calculated in the buying power of it at that time. Other prescriptions of the Code of Hammurabi regulate the wages of the ordinary workman. According to this ten shekels of silver was about the yearly wage of the ordinary workingman. In our time this would amount to six hundred dollars or more. The fee then must be considered as about equal to that amount of money in our time.

This is I think the only fair way of comparing wages and fees in our period and that. It shows that the professional man was

because of his skill often expected to make as much money in a single day as a workman would in a year. While this old-time legislation was thus favorable to the medical profession it was also very careful of the rights of patients and prevented unskilled men from imposing themselves upon people by inflicting heavy damages for malpractice. This side of the Code of Hammurabi is particularly interesting. It might seem that it would be a rather risky thing to take up an operation in any case that was at all dubious. We might even be tempted to conclude that such laws would discourage the development of surgery and keep surgeons from exercising such skill as they had, on cases where there might still be a hope of benefit though the chances were against ultimate cure. It is very probable, however, where men had thoroughly established reputations, such damages were not imposed on them for deaths that were evidently almost inevitable without their services and that the law served only to prevent the unskilled from intruding themselves unwarrantedly into the performance of operations that by law were so lucratively rewarded.

"218. If a physician operate on a man for a severe wound with a bronze lancet and cause the man's death; or open an abscess (in the eye) of a man with a bronze lancet and destroy the man's eye, they shall cut off his fingers.

"219. If a physician operate on a slave of a freeman for a severe wound with a bronze lancet and cause his death, he shall restore a slave of equal value.

"220. If he open an abscess (in his eye) with a bronze lancet, and destroy his eye, he shall pay silver to the extent of one half of his price."

Among the Greeks the doctor's honorarium as it is called in many of our foreign languages now, was literally honorary inasmuch as there was no fixed fee and payment was by presents. This was entirely the case in older times and in later times conditionally so. The doctor's reward was still a present though he had something to say about the value of it. An old Greek legend, probably earlier than 1000 B. C., tells that Jupiter struck one of the old physicians by lightning because he had set a money price upon his services. By the time of Hippocrates the taking of

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\*This is about the ratio of indemnity for the loss of an eye that the insurance companies have apparently agreed upon in the modern time, that is about half the full value of the accident policy.

money, however, was considered professional though we have no definite records of the amounts paid for various purposes. Special arrangements were made in the Grecian cities for the medical care of the poorer class by physicians somewhat as in our municipal dispensaries, only the physicians who gave their services to them received a definite salary. From this salary we can judge reasonably what medical services were thought to be worth. Regular dispensary attendants received from \$300 to \$500 at a time when this was four or five times as much as the yearly wage of a workman.

Some of those who were sent for on special consultations by Kings and Emperors received very large rewards. Demokedes received from Darius, the King of the Persians, for reducing a dislocation, a pair of gold bracelets. When he did not seem to be quite satisfied with this reward the King gave him further a handsome fully furnished house and the right to sit at the table with the King. Cleombrotus is said to have received for the successful treatment of Antiochus I a hundred Attic talents. This would be equal to over \$100,000 in our money.

It is well known that among the Greeks of the classic period the honorarium was regulated according to the wealth of the patient. The maximum seems to have been as high as \$100 per visit while the minimum was a drachma, which would be about twenty cents. Aristophanes has an even lower figure as the minimum and suggests that physicians often had to take absolutely nothing. That, however, is nothing new. The satire is easy to understand and is quite as applicable in our own time as to the Grecian times. What the regular charges were for the different classes of citizens we have no means of determining. The Grecian cities were democracies and the laws could not be so drawn in the kingdoms as to indicate what the different classes should pay, since every man was supposed to be as good as any other, and special arrangements had to be made according to the value of the physician's services to particular patients.

In Rome we have not many details of the compensation paid for ordinary medical services. We know something more about the large fees that were given on special occasions. Many of the physicians at Rome, indeed most of those who reached distinction

during the days of the Empire, were Greeks. Rome's literature was an imitation of Greek, and Roman science and philosophy were largely adopted from Greece. "Greece conquered her conquerors" is the brief formula for this phase of history. Galen for instance, who practised at Rome among the high officials at the end of the Second Century of our era, received many large fees. From the Consul Boethius for instance, for the successful treatment of his wife, Galen received the equivalent of \$2000 in our time. This was apparently the honorarium for bringing her through one of the acute illnesses, probably of a month or more in duration. Before this time there are some stories which serve to show that Greek physicians in Rome must have made very large sums of money. Mommsen, the great German historian of Rome, has given a detailed account of two brother Greek physicians at Rome in the first century of the Empire, whom he takes as types of the tendency to accumulate a fortune in medical practice at Rome and then bring it back to their native city in Asia Minor to erect magnificent buildings or endow institutions.

One of these is Xenophon, son of Heraclitus of Cos, well known from Tacitus (*Ann.* xii, 61, 67), and Pliny (*H. N.* xxix, 1, 7), and from a series of monuments of his native place (*Bull. de corr. Hell.*, v, 468). As physician-in-ordinary (which title first occurs here) to the Emperor he acquired such influence that he combined with his medical activity the position of imperial cabinet-secretary for Greek correspondence, and he procured not merely for his brother and uncle the Roman franchise and posts as officers of equestrian rank, and for himself, besides the dignity of a knight and the rank of officer, the decoration of the golden chaplet and the spear on occasion of the triumph over Britain, but also for his native place freedom from taxation. His tomb stands on the island, and his grateful countrymen set up statues to him and to his, and struck in memory of him coins with his effigy. He it is who is alleged to have put an end to Claudius, when dead-sick, by further poisoning, and accordingly, as equally valuable to him and to his successor, he is termed on his monuments not merely, as usual, "friend of the Emperor" but specially friend of Claudius, and of Nero (so according to a certain reservation).

His brother, whom he followed in this position, drew a salary

of 500,000 sesterces (\$25,000), but assured the Emperor that he had only taken the position to please him, as his town-practice brought in to him 100,000 sesterces more. In spite of the enormous sums which the brothers had expended on Cos, they left behind an estate of 30,000,000 sesterces (\$1,500,000).

The elder Pliny tells us of two distinguished Court Physicians Stertini by name whose professional income was estimated at nearly the equivalent of \$25,000 a year. Though their names are Latin it is not unlikely that they were of Greek origin for they were born in Naples and that portion of Italy was called at this time Magna Graecia. These two physicians devoted their ample fortune to various benefactions for the City of Naples.

The ordinary physician in Rome seems to have asked the equivalent of about thirty cents in our money for each visit. With the purchasing power of money at that time, however, it is probable that this amount was equal in value to about the ordinary fee of the modern time. The middle classes lived rather simply at Rome, though the very wealthy made their money just exactly as our own great fortunes have been made,—by the opportunity to collect taxes for a government and the chance to corner provisions and monopolize various forms of industry and trade,—developed very luxurious habits. It was among these that the Greek physicians particularly had the call and received such large fees. Hence the tradition, that in spite of the large salary offered as Imperial Physician one of the Xenophons declared that he could make even more on his private practice.

Fees during the Middle Ages were maintained at a good professional standard. This is what we might expect from what we now know of the status of medicine during the period when the universities were gradually acquiring their prestige and drawing the large numbers of students that attended them. Knowing what we do of the universities we should be surprised had their medical schools been of low rating, but it is almost as much of a surprise to find how high were their standards. According to the law of Frederick II, issued for the Two Sicilies, the southern part of Italy and Sicily proper, three years of preliminary study at the university were required before the study of medicine might be taken up, then four years in the medical school itself

and an extra year of practice under the direction of a physician before a license for independent practice was issued. An additional year of study at anatomy and surgery were required if a physician wished to take up the practice of surgery. It would have been possible to maintain any such high standard as that only by having fees that would in some way commensurately reward the seven or eight years of preparation required. We know from charters of the medical schools of the universities issued by the Popes that practically all the schools were required to maintain these standards. This same law regulated physician's fees.

According to the wording of the law, which I translated for the *Journal of the American Medical Association*, February 1, 1908, the physician was required first of all to give his advice to the poor without asking for any compensation, and besides to assume the duty of reporting apothecaries whenever it came to his knowledge that their drugs were harmful or of less than normal strength. He was supposed to charge his patients by the day and to visit them according to their needs. They were a little demanding on a physician's time in this respect, but the professional fees made it worth while. Evidently the purpose was to require the physician to visit his patient often enough for the patient's good, and yet not allow him to be tempted to visit oftener than was necessary for the purpose of running up his bill. In many ways there was practical wisdom in the arrangement. The law said:

"A physician shall visit his patient at least twice a day and at the wish of his patient once also at night, and shall charge him, in case the visit shall not require him to go out of the village or beyond the walls of the city, not more than one-half tarrene in gold for each day's service. From a patient whom he visits outside the village or the wall of the town, he has a right to demand for a day's service not more than three tarrenes, to which may be added, however, his expenses, provided that he does not demand more than four tarrenes altogether."

Once more in order to understand what the fees were worth we have to take not only the value of the money in terms of our money, but the buying power of it in our time.

A tarrenus or tarrene in gold was equal to about thirty cents of our money. Money at that time had from fifteen to twenty times the purchasing power that it has at the present time. An ordinary workman at this time in England received about four pence a day, which was just the price of a pair of shoes, while a



fat goose could be bought for two and a half pence, a sheep for one shilling two pence, a fat hog for three shillings, and a stalled ox for sixteen shillings. (Act of Edward III fixing prices.) At the present time with our wages much higher, it is quite impossible for the working man to buy a pair of shoes for less than two days wages. As for the other necessities of life, they absorb so much money as to make our high scale of wages quite low compared to those that were paid in the Thirteenth Century. When a fat goose can be bought for a little more than half a day's wages there is no danger of the workingman lacking meat. Other meats are sure to be in proportionate price to this and such they actually were. Fifteen cents does not seem much for a professional visit, but when we recall that it is twice a laborer's daily wage it is the equivalent of from three to five dollars in our time.

Some of the fees of the Middle Ages as we have obtained information with regard to them show that the question of professional reward, and not of anything like payment for services rendered, was the uppermost thought. Men paid not so much according to what was done for them as according to the value of the services rendered to themselves. In the Fourteenth Century John Ardern, the famous English surgeon, whose text-book of surgery is yet unprinted (we have a fine copy of it in the Surgeon-General's library), had a special reputation for the operation for rectal fistula. His regular professional charge for this was at least one hundred gold sols. The sol was an old French coin, the twentieth part of the old French livre, equivalent to twelve deniers. The word soldo comes from the same root and these are of course small coins. Even 300 years ago Ben Jonson in "Volpone" said of one of his characters "for six sols more would plead against his Maker." Six sols was the expression for next to nothing from which apparently we get the tradition for our juries to give six cents damages. The gold sol of the Middle Ages, however, was a very different coin and a rather valuable piece of money. It was probably worth not less than five dollars in our values. Ardern's charge of one hundred gold sols therefore, would be a rather good fee even in our time.

Curiously enough, especially for those of us who are accustomed to think of progress as being continuous, there was some setback in medical education and in professional standards toward the end

of the Fourteenth and the beginning of the Fifteenth Century. These were due to social disturbances, to wars and to the loosening of political and ecclesiastical authority in many parts of Europe. Even with this, however, there was not such a descent in the scale of fees as to make them less than professional reward or merely wages for work done. Prof. Puschmann, in his *History of Medical Education*,<sup>4</sup> discusses the fees that were paid in the various cities of northern Italy and shows that they continued to maintain the standards created by the Emperor Frederick's law about the middle of the Thirteenth Century. At this time the number of physicians had largely increased as a consequence of the ease with which University medical education could be obtained, so that the fees mentioned are an index of high professional status.

For each professional visit in ordinary cases at Venice in the Fourteenth and Fifteen Centuries 10 soldi were paid. In Milan the pay was more like that on the scale of the older time according to the days illness of the patient. For every day of treatment from 12 to 20 soldi were usually charged. For a night visit the charge was a ducat. If the physician was called outside the city, however, his charge for each day was from four to six lira. Soldi are of course small coins. They represent the pennies of the modern time. It must not be forgotten, however, that money had very different values, meaning by that, buying power in the olden time. The workmen of the middle ages got by law but eight cents a day, as I have said, but for that he could buy more than the workman of our time with his daily wage. The maximum prices of the necessities of life were determined by the same law which regulated the minimum wage that might be paid in wages. When physicians got from ten to twenty cents a visit then, we must calculate the value of that in buying power in our time. In Shakespeare's time, at the end of the Sixteenth Century, money was worth about eight times what it is at the present time. In the preceding century it was worth probably ten or twelve times as much. It is very probable that ten soldi was not less than a dollar and twenty soldi probably more than two dollars in the early part of the Sixteenth Century. Earlier the same amounts would be even more valuable.

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<sup>4</sup> Puschmann: *History of Medical Education*, English translation, London, 1893.

The ducat was a gold coin issued in various countries in Europe and supposed to contain about three and a half grammes of fine gold. This was worth about \$2.40. The buying power varied at various times but was probably not less than ten dollars at any time. The lira is worth at the present time twenty cents. A visit outside the city was supposed to be somewhat more than a dollar for each day then, at a time when money had probably not less than eight or ten times the buying power of money at the present time. To this a physician was not only allowed but was supposed to add his expenses. As a matter of fact visits outside of the city were usually paid to the nobility. Under these conditions a vehicle of some kind or an escort for horseback riding was sent to accompany the physician and his comfort and convenience were courteously provided for until his return home.

One wonders whether they had any difficulty in collecting their fees in the Middle Ages and is not surprised to find that some of the advice given to young doctors included that as to when they should present their bills. They had evidently learned the lesson at that time that people are long on gratitude when they are ill and short on payment when they are well. Young doctors are told in our time not to wait long after their attention to the patient to present their bills, but just as soon as convalescence is properly established, or if during the illness itself the regular time for sending a bill should come, not to hesitate to send it and above all to let patients understand just how much of a pecuniary obligation they are being placed under. There is a little volume of instruction for the young physician issued during the Thirteenth Century, the author of which is unfortunately anonymous, but which has been incorporated in the *Histoire Littéraire de la France*, tome 22, page 106. This author is very explicit and direct in his advice. He says "When you have brought the patient to a state of good health you must ask permission to cease your visits, lest further attendance might be a cause for shame. You should then talk to the head of the house or to those who are more closely related to the patient in the following terms: 'God the all powerful has directed our actions and has deigned to restore your relative to health by our ministrations. I hope now that he will remain in good health and that I may be permitted

to surrender him to the care of his family. If any of you should be stricken with disease and should wish to call on us we shall be ready to lay everything else aside in order to come to your assistance. In the meantime a proper remuneration for our services in the present case will be a pledge for our future readiness to help you in every way.' It will be well, therefore, if during your attendance on the sick person you have from the beginning of your calls been careful of the feelings and susceptibilities of those who are caring for them since it is well to stand high in their favor. The sick person is practically always sure to consult them as to your fee. It is always surer, however (well do we physicians all know it), to receive payment while the ailing person is not as yet entirely well. Otherwise there is always some risk of not being paid. For, a hand that is ready to give is sometimes drawn back after the cure is complete. Once you have received your fees, give due thanks, say your adieux to all and withdraw in peace."

It seems interesting to place beside these old-time laws and customs in the matter of fees an early American medical society's regulation of the same matter. It serves to show that more than a century ago physicians realized very thoroughly their professional standing and endeavored to maintain it. I have taken my discussion of the fees from the history of the New York State Medical Society which I wrote for the Centenary of that organization some five years ago. There had existed in New York City just at the end of the Eighteenth and the beginning of the Nineteenth Century a medical society calling itself The New York State Medical Society, though its members were all from what is now New York City. This Society seems to have had a rather active existence for some ten years. Scientific papers were read, the health of the city discussed, suggestions made for the prevention of epidemics, and for general municipal hygiene and sanitation and for the ethics of professional life. Practically all the subjects that are discussed in medical societies at the present time came up before this old-time American medical society. Among other things the members of the society established a code of charges for professional services. It is this table of fees that interests us here because of the chance to compare it with the past and also with the present.

Knowing something about the purchasing power of money at

the time, at least three or four times what it is now, one might expect that these charges would be ridiculously low, according to our modern standard. This proves, however, to be by no means the case. The physicians of the time had very properly a high appreciation of the value of their professional services. A hall for a meeting in a Broadway hotel, with fire and candles, might cost only two dollars a month, but the prices asked for visits were nearly as high as the average of the modern time. It is to be presumed that these were considered the highest charges that would ordinarily be made, and that while many physicians might accept less, no one would be expected to ask more. It is interesting to find that the list begins with the two items "Verbal Advice" and a "Letter of Advice," for which respectively, five dollars and ten dollars are charged, showing that the physician of that period did not consider that the principal item of value in their services was the writing of a prescription but rather the giving of advice.

In those days, of course most of the physicians carried their own drugs with them, and it might be expected that most of the drugs were thrown in for the charge for the visit. This was far from being the case, however. In fact, a reading of the list of charges for drugs will serve to show that they were quite as high as are the modern druggists' charges if indeed not much higher. Twelve cents for each powder given, and twelve cents for each pill or dose of pills one might say was quite as much as the tariff would stand. A single dose of medicine dispensed without a visit at sixty-two cents is considerably dearer than the renewal of a prescription at the drug store afforded by modern custom.

Consultations were not appreciated at as high a value in the olden times. The first visit in consultation, five dollars, and subsequent visits, two dollars, cannot but seem very small. It is rather interesting to find that mileage was charged at a dollar a mile, a price which obtains at the present moment, we believe, in many towns much larger than New York at that time. A visit to Brooklyn was only three dollars. One to Staten Island was ten dollars, and this charge was doubled in the winter time. The scale of charges for infectious fevers, where personal danger was incurred, were higher than ordinary visits, and remind us that these were the days when smallpox and typhus fever raged virulently, and when yellow fever, then considered to be an ex-

tremely contagious disease, often ravaged New York. The charges for the venereal diseases are made in lump sums for the whole course of treatment, a practice which early New York physicians doubtless found advisable from the capriciousness of such patients.

With regard to the operations, the list given shows that there was much more operating in New York at the beginning of the nineteenth century than might be expected. It is to be presumed that charges were not set down in the list for operations that were not likely to be performed, or had not actually been performed. Bronchotomy (tracheotomy) is found in the list of operations. The charges in midwifery cases can scarcely but be considered high, considering that money at that time was worth at least three times as much as now, and had, indeed, for ordinary living expenses, at least four times the purchasing power of our present currency.

The following is the rate of charges for professional bills agreed upon:

We, the subscribers, practitioners of physic and surgery in the State of New York do agree upon the following rate of charges for our professional services from and after the first day of July, 1798, agreeably to which rates we do recommend our bills to be presented every six months and oftener, if circumstances permit:

Verbal advice .....	\$ 5.00
A letter of advice .....	10.00
An ordinary visit .....	1.00
A visit with a single dose of medicine.....	1.25

#### MEDICINE TO BE PRICED AS FOLLOWS

For powders, each .....	\$ 0.12
Pills, each dose .....	0.12
Boluses, each .....	0.25
Electuary, per ounce .....	0.50
Decoctions, \$1.50-2.00 per lb., or per ounce.....	0.12
Infusions, \$1.50-2.00 per lb., or per ounce.....	0.12
Mixtures, per ounce .....	0.12
Lotions per pound .....	1.25
Tinctures, per ounce .....	0.25
Vol. Spt., per ounce .....	0.50
Ointment and cerate, per ounce .....	0.25
Blistering plasters, according to their size, from \$1.25 to .....	2.00
Other plasters from 50 cents to .....	2.50
For a single dose of medicine dispensed without a visit..	0.62

## CONSULTATIONS

The first visit in consultation .....	\$ 5.00
Each subsequent visit in consultation.....	2.00
A night visit .....	5.00
Visit at a distance from town per mile.....	1.00
A visit to Brooklyn .....	3.00
A visit to Pawles Hook (Paulus Hook) .....	5.00
A visit to Staten Island .....	10.00
(The last two charges to be doubled in winter or in tempestuous weather.)	
The first visit in epidemic fevers, or in other cases where personal danger is incurred .....	5.00
Each subsequent visit under these conditions.....	2.00

## CHARGES

For curing a simple or virulent, gonorrhea, from \$10.00 to \$20.00	
For curing confirmed syphilis, from \$25.00 to.....	100.00
For dressing a blister, from 50 cents to .....	1.00
For dressing wounds, from \$1.00 to .....	2.00
For applying cupping glasses .....	4.00
For bleeding in the arm .....	1.00
For bleeding in the foot .....	2.00
For bleeding jugular vein .....	2.00
For opening an artery .....	5.00
For attending in smallpox from \$5.00 to .....	10.00
Scarrifications of the eye .....	5.00
Punctures in the Oedematous Swellings .....	2.00
Inserting a Sprue .....	2.00
Inserting a seton .....	5.00
Introducing a catheter first time .....	5.00
Introducing a catheter each subsequent time.....	2.00
Extracting a calculus from the urethra .....	10.00
Reducing a simple fracture, from \$10.00 to .....	20.00
Reducing a compound fracture .....	30.00
Setting dislocations, from \$5.00 to .....	20.00
For reducing a polpus ani .....	5.00
For reducing hernia .....	25.00
Opening an abscess, from \$1.00 to .....	5.00
Amputating the breast .....	50.00
Amputating the arm or leg .....	50.00
Amputating the joint .....	100.00
Amputating the finger .....	10.00
Amputating the penis .....	20.00
Extirpating the eye .....	100.00
Extirpating the tonsil .....	25.00
Extirpating the testicle .....	50.00
Extirpating a polypus .....	25.00
Perforating the rectum, nostril or urethra.....	10.00

Paracentesis of the abdomen .....	10.00
Paracentesis of the thorax .....	50.00
Operation for an aneurism .....	100.00
Operation for harelip .....	25.00
Operation for hydrocele .....	25.00
Operation for hernia .....	125.00
Operation for fistula in ano .....	50.00
Operation for fistula in mundo .....	25.00
Operation for phymosis .....	10.00
Operation for paraphymosis .....	10.00
Fistula lachrymalis .....	25.00
Wry neck .....	25.00
Cataract .....	125.00
For operation of Lithotomy .....	125.00
For operation of Bronchotomy .....	25.00
For operation of trepanning .....	100.00

MIDWIFERY

For a common case from \$15.00 to .....	25.00
For tedious or difficult cases from \$25.00 to.....	40.00

Just one thing this review of fees for some five thousand years makes clear, it is that at all times physicians have received professional compensation, that is, a reward for services done, not according to the time or effort required but the value of the benefit conferred on their patients. As a rule patients have been willing to pay not only this but to add honors and distinctions of other kinds.



# Postgraduate Course

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## WOUNDS BY FIRE-ARMS\*

BY WILLIAM S. WADSWORTH, M.D.

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THE term gun-shot wound has come to be accepted as an inclusive one and has until very recent days served well to convey the general impression of all wounds produced by the discharge of any sort of gun. Very recently rifle experts have objected to having their own instrument called a gun and certain literature on pistol-shot wounds has still further discouraged us from using the term. I am therefore constrained to use an older term which will I trust serve us for a time, at least.

It would seem that the subject of wounds by fire-arms had been sufficiently written upon to be more than completely exhausted. Fire-arms of various sorts have been used for more than five centuries and the literature of the subject is simply enormous, both on the theory and art of their use and on their results on the body.

In spite of it all we are face to face with the grossest ignorance of the fundamentals of the whole subject and this is evident in nearly every paper and book, from the reports of the ordnance department of the army to the most florid review of cases by the young resident physician who has just treated a case.

For more than ten years I have been studying gun-shot wounds in the human body and I have seen hundreds of cases. Incidentally I worked hard during more than ten summers on ranges with rifles and revolvers. I have gone over masses of literature and much of

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\* This is the subject for the June 2, 1911, meeting of the Postgraduate Course of the American Medical Association, as planned by the Warren County Medical Society, Bowling Green, Ky., through a special committee of which Dr. J. H. Blackburn is the chairman.

it had better never have been written. Worse than all else I have met experts; judges who shot at frogs when they were boys and therefore know more than all the rest of the world; army experts, who are almost as ignorant and fully as absurd and who propagate error with a clear conscience and will never learn that reduced charges do not act as they mathematically should; police officials who insist that a bullet gains velocity after it leaves the gun; writers of monographs who are unable to handle a foot rule intelligently and surgeons who read a half a dozen works and after diligently collecting the errors of all of them write learnedly in text-books. I am going to get away from all such and point out a few simple things about fire-arms and wounds by them. You will find small parts of what I say scattered through the literature. Some things you will not find anywhere, and the whole is the result of close observation and hard thinking. The first thing to study about fire-arms is powder, the next thing is the tube that holds the gases generated by the explosion and directs the projectile. Then the projectile thrown by the combination and, then the damage done by the combination, not simply by the projectile. After that we study all the thousand and one things that are associated with the discharge of the gun and we have a medicolegal case. It is absurd to talk learnedly about powder marks and wounds of entrance unless we have a rather clear conception of how powder acts and the principles and laws of projectiles.

The ordinary powders that we are likely to meet with are grouped into two great classes, black and smokeless. The old black powders that were known as gunpowder, made of charcoal, sulphur, and nitre, exploded on being lighted, giving off fumes of sulphur dioxide and residues of potassium, sulphur, and carbon. They were often carelessly made and black carbon smoke was left. They were ground and dried and often burnished and sometimes coated with graphite. Every process they were put through gave a chance for variation in method and in result. The powder marks on the body in a medicolegal case must be interpreted in the light of a knowledge of the powder used. A fine grained, burnished powder will not throw unburned powder grains as will a dirty, caked mixture of fine and coarse powder. You cannot tell how far away a revolver was when fired simply by applying the pleasing generalizations

from a set of experiments where one powder and a very short-barreled revolver was used. Big grains of powder carry further than small ones—that is very simple, but how often is it remembered? One cartridge has the powder jammed in so tight that it simply cannot ignite evenly and freely—such cartridges throw a projectile feebly but cause fine flash and flame and enormous powder markings. The grains of powder that do not ignite are carried out of the barrel as projectiles like shot from a shotgun, and the rush of hot gases deflects the little grains so as to give a curiously shaped figure when we try to plot them all, something like a spindle, wide at one end and tapering at the other, to again dilate into a sort of second cone of expansion. I have had diagrams made that will roughly indicate what I mean (Plate I). The main mass of the gas tends to move along the line of the axis of the barrel, as a sort of column; by the expansion from the relief from the restraint at the muzzle it spreads rather suddenly in all directions but the column is maintained by the remainder, whereas the gases that spread about do not go far to the side and the powder grains quickly lose their momentum after leaving the central gas column. The projected powder grains may ignite (explode) at any time while within the flame area, even after leaving the muzzle, but after that only by accident. The hot gases rushing out of the muzzle have an enormous power, and if the muzzle be pressed against an object firmly enough the barrel may explode, but if pressed against the surface of the body the column of hot expanding gases will cause a frightful wound under the skin. When the powder charge is considerable, as in shot-guns, the gas wound is often more serious than the shot wound when the muzzle is pressed close. If during the passage of a bullet through the skull there is delay owing to resistance being overcome and the velocity of the bullet suddenly drops the gas tends to rush on and form a pocket between the skin and the bone. I was called some years ago to one of our large neighboring cities to determine whether a wound was accidental or suicidal, and finding a large sized gas pocket under the skin I had no hesitation in saying positively that the muzzle had been pressed firmly against the temple. I have often been surprised at the lack of attention this subject has received; this gas pocket is seldom found where the progress of the bullet has not been

checked. It must not be confounded with the explosive effect of high velocity bullets on such organs as the liver, but it may occur at the same time, and I know of a case where the gas wound of the liver and the ripping of the adjacent tissues was astonishing; practically all the gas had entered through the abdominal wall; about half had ripped up the liver and the rest had blown out on all sides, destroying and burning the peritoneum and the ligaments.

Where the muzzle is only moderately pressed against the skin we find a tendency of the gases to burst the skin outwards, giving a large ragged wound which is not a wound of entrance, though always so called, but a wound of exit of the gas. If the delay of the bullet is marked and the muzzle quite firmly pressed in then we may have a gas wound of very considerable size, larger than the muzzle in fact. The gas column that follows the bullet is not only powerful itself but it acts as a sand blast, carrying powder grains of the smaller size and debris and giving an eroded surface in close-range wounds. This common phenomenon is usually slurred over in the literature and confused with the true burn, which usually occurs with it. The great heat of the gas of course gives rise to the usual singeing of hairs or clothing near the wound and at times burns the skin itself severely, the extent of these burns depending on the powder, the condition of the things burned, and the distance from the muzzle. All gas phenomena require very close range, usually less than 18 inches. Within that distance we have another result that is often confused with the burning, the smudge, which is due to the debris and smoke from the powder. It appears to accompany the after-coming gas, and where there is flip usually is found higher than the burn and where there is no flip (vise-fixed guns) it appears to mask the singe. This smudge changes on exposure to the air in experiments where paper has been used and resembles the fouling; it is in fact the same thing. Probably the lubricant used in most low-power cartridges plays a large part in the composition of this. The powder grains having been driven out with considerable force may cause serious wounds. How far they can be carried is debated. Probably a little experience and thought would do away with such debates. The safest way in any given case is to use the same ammunition and the same gun and under similar conditions carefully measure the dis-

tances to the intercepting screens and then definite conclusions will be safer. This gas and smudge not only go out of the muzzle, but the mixture escapes wherever there is a crevice and wherever it goes it leaves a mark. In revolvers this is often of the greatest importance, for there is usually quite a gap between the back end of the barrel and the front end of the chamber. Through this gap smudge escapes freely. If the hand of the person who fired the pistol shows this smudge it is often of great importance. (Plate II.)

I was called in to investigate a case where a family was trying to collect an accident insurance on the life of a suicide. I was able to point out this smudge on the forefinger of the right hand, so blown into the lines in the skin that washing had not dislodged the mark. The invented theory that the revolver had gone off as it struck the floor was thus destroyed. A man was suspected of shooting his wife, but the free smudges on the inner side of her hands, where they would be if she had clasped the revolver with both hands as it was discharged, completely freed the man from suspicion. In another case a man claimed his wife had shot at him and then shot herself. The discovery of smudge on the cartridges in his possession gave the clue which led to clearing up the case.

In close range shots the smudge coming from the barrel late allows time for the flip to accumulate and helps in determining how the weapon was held. Many good rifle and revolver shots who have practical experience but little or no knowledge of mechanics deny that the flip affects the bullet. The law that action and reaction are equal and in opposite directions and at the same instant applies to fire-arms, and I have taught a number of men to hold a revolver so as to minimize the flip. In a properly held revolver the flip is up, or toward the hammer side, but there are other directions which it may take. It was a long time before I learned how to overcome a serious flip to the left in shooting a large 0.38 S. & W. revolver. Most of the experts denied the flip, my wrist was small and very free but inclined to be a trifle flexed from long use of instruments, and as a result the handle was always to the left of the axis of the forearm. (Plate III.) The flip affects the singe mark and the smudge about a wound very materially. Generally it will be toward the hammer-side, but often this will be very materially modified by the grip on the handle at the instant of discharge.

The things we have gone over must be slightly modified if the new smokeless powders have been used. (Plates IV and V.) These are generally mixtures of a highly explosive substance with modifiers so as to regulate the rate of explosion. They all depend on the unstable nitrites for the real power. Most of them will burn rapidly if ignited and require some form of detonation to explode. They give out various compounds of nitrogen which are more or less corrosive and the regulating substances are found in the residue. In order to have the particles explode a cap is detonated so as to flash over them in the cartridge. To facilitate this the powder is made into many different forms, the commonest being granules, flakes, beads, and tubes. The gas-burn and singeing will be as in the black powders. The smudge is usually quite different, being often orange-red and usually less. The powder grains may ignite but not explode outside the barrel and so produce different types of powder marks, depending on the size and shape of the grains. The charges contain less powder and fewer grains than in black powder, so that with equal velocities, there will be less chance of powder marks. Good powders suitably selected for length and strength of barrel are largely burned within the gun, but in ordinary cases there is little or no adjustment of charge to pistols and the effect gotten by tests made with a 2¼-inch barrel will hardly hold in a 6-inch barrel.

While black powder can be kept for years and be in excellent condition the smokeless powders are very apt to deteriorate and most of them weaken rapidly even when sealed up. Old cartridges were but poorly crimped and dampness often got inside, and in some old copper shells of French make the powder hardly expelled the bullet from the gun. Old black powder may be good or bad. The only way to be sure is to test it. Some smokeless powders are loaded with graphite and produce a nasty smudge. Gas wounds are about as with black powder but show less smudge.

For medicolegal purposes what we have gone over is of utmost importance; for the surgeon the most important is the projectile and its wound. The projectile in all modern small arms is chiefly of lead, which may be hardened or cased in some sort of jacket. Round shot are often of hardened lead. Formed bullets are of lead but may have a small quantity of tin or some other metal added. Where high velocity is to be attained it is necessary to protect the

lead core with a patch or jacket which enables the grooves and lands of the barrel to set the bullet spinning without stripping off the lead. The common compositions of these jackets are mixtures of copper and nickel, plain copper, and alloys of iron. While the tensile strength is much greater than lead they do not prevent the bullet from going to pieces or losing its shape when it hits a hard object such as dense bone.

A very common mistake of writers is to confound a soft copper-nickel jacket with a high-tension steel case, and proceeding to reason on the basis of steel to proclaim wonders about the new bullets that are very far from facts. The jacketed bullet for all velocities above 1000 ft. per second and for distances over 1500 ft. appear to have decided advantages for the marksman, so we may expect them to be used more and more. The projectile remains bright and clean longer under ordinary conditions and the surface is less easily marred but under certain conditions they corrode. When travelling over 2000 ft. per second they will hold together enough to penetrate any ordinary human structure without breaking up, but as the velocity drops (the penetrating power goes down much more rapidly than the velocity, say as the square root) there comes a point when the resistance is too much for the momentum of the bullet to overcome quickly and then the bullet piles up on itself just as it does when it strikes any very hard object, and the lead crowds to the front part of the bullet till the nose of the jacket bursts. When this happens the wounds are very serious and have given rise to rumors of the use of "dum dum" or soft nose bullets or of explosive bullets. This bursting of the jacket is said by competent eye witnesses to cause more serious laceration than the old lead bullets. This deadly bursting quality has been reduced by the reduction of the amount of lead in the core and the sharpening of the nose. This sharpening of the nose has greatly increased the penetrating power which also lessens the chance of bursting. But for medicolegal purposes the lead bullet will be the stand-by, for few indeed are the cases of murder where fancy fire-arms with jacket bullets have been used. Crime is apt to be clumsy and crude, and cheap as well. But because of the occasional cases where information is sorely needed I have included them, and also because so much of the information that is to be obtained is bad. Most medicolegal cases of wounds by

firearms will be by lead bullets. Occasionally the paper wad or a piece of the metal shell of a blank cartridge will cause a fatal injury. I will not readily forget my search for the bullet in a case of a boy who was shot by another boy on a Fourth of July. After removing a little dark hard object from what appeared to be the bottom of the wound I returned to the search for the bullet. I was much annoyed at not finding one until I made a careful examination of the hard object which proved to be a compacted rolled up piece of the copper shell of a 0.22 blank. This accounted for the peculiarities I had noted in the wound of entrance and was confirmed very satisfactorily by positive information that neither of the lads had had anything but blanks in their possession.

The little 0.22 caps in toy rifles have furnished a number of tedious hours' work, for the bullets are so small that great care is required in following their paths and recovering them. Now and then the wad of a shot-gun causes fatal injury, but usually it is associated with the load of shot. I have seen cases where the wad appeared to have helped very materially in causing the fatal penetration in chest wounds.

Shot wounds which are not fatal are quite common and need little comment though their study materially helps us to grasp the factors in severe cases. Each shot has a penetrating power depending on its mass and on its velocity. Each shot makes a limited area tense before penetrating. If these areas overlap the chances of both or either are greater; the greater the number of overlappings there are the greater the likelihood of severe lesions. If, as is always the case, the shots arrive at different instants a sort of re-enforcement in the tension may result before the surface recovers its normal resistance. At the point where the mass of shot strikes, in short range cases, there is little question of how the penetration occurred, as there is a complete destruction of all tissue, but when the distance is sufficient to insure considerable spreading of the central core of the load and where it is desirable to estimate the distance from the muzzle these details become of vital importance and they can only be used cautiously, and after taking into account the sort of barrel and the best obtainable information as to the charge of powder and shot, all of which modify the results. The close range wounds from shot-guns often furnish excellent examples of gas wounds within the body and shot wounds on the surface.



The not uncommon method of committing suicide by placing the muzzle in the mouth and pulling the trigger by a string attached to the foot causes an explosion of the brain case that can be accounted for only by a knowledge of gas wounds. Rarely do we see wounds made by round bullets except from shot-guns but now and then there is a case of suicide by means of an old-fashioned powder-and-ball pistol or a case from the country where an old smooth-bore rifle has been turned on the victim.

I have little experience in wounds made by large round balls, but from the accounts by the best writers it would appear that round bullets wander from the straight path more often than do elongated ones and that they roll about easily following tense fasciæ even in curved lines. They have much less penetrating power and cause greater bruising and destruction of the tissue, because instead of going through they give up the energy of their momentum to the tissue thereby causing wide-spread concussion. They cause greater injury to vessels in that they less readily push them aside in passing. As they are practically never thrown at high speed their action is more like that of spent bullets that have lost most of their initial velocity.

Of the conical or conoidal bullets there is an appalling variety. The reasons why we are obliged to study the types, at least, are so that we can be sure of our conclusions as to the identity of the bullets found and the conditions of firing. As an illustration, a cheap German cartridge was found to be associated with a number of crimes. No reputable dealer knew anything about it. After months of searching a pawnbroker, who was a witness in a case, told me where he purchased them—the bullets were marked in a peculiar way and were, with other associated peculiarities of these cartridges, of value in subsequent murder trials.

A glance at the figures in Plates VI and VII will show the marked differences of common types of bullets. The calibre is by common habit the way of designating a bullet, but this is often very misleading and a little display of accurate knowledge in a court got me rather severe censure from a very ignorant and irritable judge. What is called a 0.38 may be 0.34 or 0.39, usually it is about 0.35. A 0.22 may be 0.205 to 0.23, but they vary less. The police constantly bring me cartridges with the assurance that they were made

by Smith & Wesson and they seem annoyed if I suggest that 0.32 S. & W. refers to the calibre. European makers use the metric system, but they make cartridges for S. & W. standard sizes.

Some makers work on the theory that a long bullet is more accurate while others work on the practical basis that the less lead and brass the more the profit, so that even in makes that are reputed as good the standard sizes vary.

It may be said that there is little variation in the size and weight of the same brand from any one house. Thus U. M. C., W. R. A. and Peters may differ from each other slightly in 0.32 S. & W. but the cartridges in a box of either make will show a remarkable evenness.

Shot varies in the same way, so that it may at times be possible to determine the make. Thus Greener gives the variation in No. 1 shot as from 69 to 104 pellets to the ounce. Calibre is to be measured with care, as a great deal depends on skill, and while it is very simple to put the micrometer on a bullet it requires judgment to avoid some very disastrous errors.

A lead bullet is very easily distorted and a 0.38 Colt may if upset either in shooting or by mishandling measure the same as a 0.38 S. & W. though the 0.38 S. & W. will not go in a 0.38 Colt pistol. The weight of the bullet varies with the calibre and it varies with the maker. One 0.22 may weigh as much as a 0.32 of another type.

The loss in weight of clean bullets by being fired through clean barrels is extremely small. Much literature has resulted from dirty barrels and not a little caused by melting of wax. The rusty barrel makes a very good file and the wax lubricant often disappears from bullets. The groove in which the lubricant lies is often the best means we have of identifying the make of the bullet as the bottom of this groove is milled and the lines may slant or end peculiarly or be wide apart. The cup at the base and the shape of the nose as well as the mark where the shell is pressed (crimped) into the bullet to hold it fast as well as make it tight against moisture all tell their story. The relation of the size of the bullet to the size of the wound has caused much bitterness and more literature, most of which would have escaped us if the author had learned how to measure even decently well. I have seen hundreds of bullet holes but have yet to see one smaller than the bullet. The question of how to measure a hole in any tissue must be

solved by the application of common sense and skill. A mouth that was measured as some writers measure bullet wounds would afford enough literature for all future generations, for when it was puckered it would measure so little and when it opened it might be very large. The lumen of a bullet wound, like other openings in the body, is best measured by a graduated cone with a gentle constant tension and at least a small amount of the very best instrument for such things—mind.

The length of the bullet is changed in firing. It tends to shorten in the barrel and it can be shortened by striking clothing before entering. The marks in such cases that appear on the nose of the bullet are often of very considerable importance. A case came under my notice where it was claimed that the bullet came from the outside of the house. The underclothing and body showed no evidence of close range. The bullet nose, however, showed a different cloth from the underclothing, and on search the coat was found and on it marks of close range.

In another and quite famous case where a man was shot down in cold blood, it was claimed that the pistol went off during a scuffle. The thin night clothing showed no powder marks and by the shape of a wound in front the wound had been of certainly not close range and with the dirt on the wound of entrance in the back and an exact correspondence between the bullet and the crescentic cut in the cloth in front, taken with the condition of the nose of the bullet, it became clear that the statements of the guilty party were false and the defense had to be changed, for which the lawyers were very viscious. The other changes that take place in the bullet are such as could easily be worked out mechanically. I have selected a number of cases for Plate VII where there have been a number of bullets recovered from the same body and where each bullet had a different story to tell. They will bear close study with the notes that appear with them. I have only twice cut out true glance shots though many times the plea has been made that the shooting was accidental and the bullet glanced. When a lead bullet glances there is always something on the bullet to show it. This is to be expected from reasoning but it is shown practically every day on every range in the country (even where jacketed bullets are used the bullet shows the mark of the glance). Anything that changes the shape of a bullet changes the wound it makes.

Sharpening the nose, as is sometimes done by negroes, makes the penetration greater, provided there is nearly equal weight. Cause the bullet to turn and the wound will show it; now and then a bullet will be so turned in the body that it will continue to move side on instead of point on; it will then cause what is called a keyhole wound.

At times we find a bullet passing through a limb before it enters the body, and in such cases it is often instructive to compare the different wounds. The wound conforms to the shape of the bullet as presented. A conical bullet coming point on makes a circular hole which of course will yield to surface tension as would a stab wound and appear like a broad slit by reason of the drawing out of the sides. If the conoidal bullet impinges at an angle so that the tangent and the surface coincide the wound will be elongated and the edge will be quite different in different parts; one side, that directly under the bullet, will show greater compression. If the shot is a glancing one one side may resemble a wound of exit while the other is like one of entrance only it is less characteristically so. An ordinary wound of entrance ought not to be mistaken, but at times anyone, no matter how expert, has a right to hesitate until he has completed his examination of the whole wound.

I remember a clean wound of entrance where the skin was thin and weak and where the fat was soft and did not support the skin, where there was no depression of the edge and no purple areola and the fat particles protruded, where it would have been improper to say off-hand whether the bullet went in or out. I also remember being sharply challenged by a learned judge for venturing to state the direction in which a bullet had gone through a man, but after I had detailed the conditions as I had found them—*areola*, depression, fine whitish fringe of tissue at entrance, fragments of rib driven into lung, fragments of spine driven into back of other lung and intercostal spaces and a ragged hole from the smashed bullet in chest wall—the case was convincing even to a rigidly legal mind where all matters of insight are strictly anathema. The direction taken by bits of clothing, fragments of bone, teeth, hair, and ornaments and the damage done are often of the greatest significance. I once spent a busy hour over a necklace that had been forced into the tissues in front of a bullet. The direction from which the bullet

had come was very hard to determine. What can happen to tissues as the result of bullets passing into and through them would far transcend the limits of our present discussion.

Anyone who hopes to make even a fair attempt at mastering gun-shot wounds must learn the resistance, elasticity, vascularity, and internal architecture of the parts of the body. This is not very difficult in a general way but where the architecture is complicated, as in the bones of the head and the structures about the throat, it is extremely difficult.

Wounds of the viscera alone would fill volumes, because every detail of the anatomy and physiology of the part involved must be held in mind before a true understanding can be reached. The errors of treatment of these wounds that I have known of would, if I were to detail them, bring me a host of brilliant enemies.

The study of death by shock, hemorrhage, infection, following gun-shot wounds, as well as the sequelæ, paralysis, constrictions hernias, pathologic processes, are all intensely interesting and practical, but I set out to say a few things that I personally know, after years of study, that were not collected elsewhere, if indeed they were to be found in print at all. I have purposely avoided going into detail where the ordinary text-books are moderately satisfactory, and have avoided more than a passing glance at the military side. In fact I have been very brief for so large a topic, but while I am conscious of briefness and omission others have omitted more and possibly with less good reason.

#### EXPLANATION OF PLATES

PLATE I.—Fig. 1, *a*, shows a diagram of a discharging rifle. A spindle close to the muzzle represents the shape of the flame discharge. The dotted lines represent the projected powder grains, roughly indicating the general shape of the discharge. The larger spindle, faintly indicated, represents the area of smudge or fine debris carried by the outrushing gas. *b* shows the section of a wound made beyond the smudge zone. Fig. 2, *a*, shows a similar discharge when the skin is close to the muzzle, showing the reflection of the gas. *b* shows a section through the wound made with erosion of lips by the sand blast and the powder grains imbedded. Fig. 3.—A similar discharge when the muzzle is pressed against the skin. The gas expanding where it finds least resistance, *i.e.*, the subcutaneous tissues, causes a typical gas pocket. If the bullet penetrates the skull then the gases will follow it so that no singeing, powder marks, or smudge will be observed on the outrush. If not the skin will be burst out by the exit of the accumulated gas, giving a large rough hole.

PLATE I.

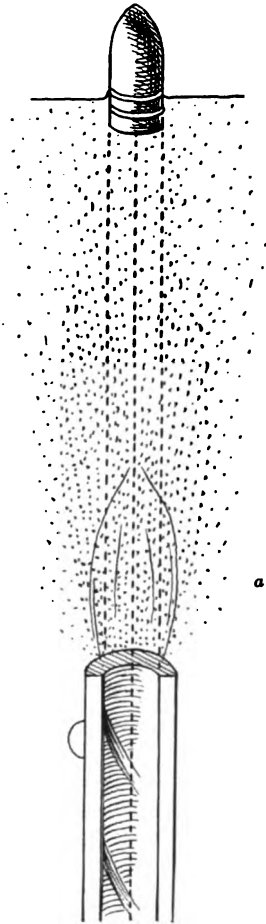


FIG. 1.

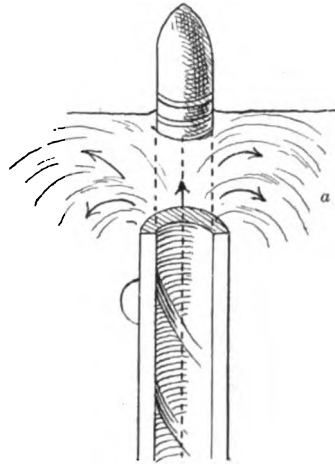


FIG. 2.

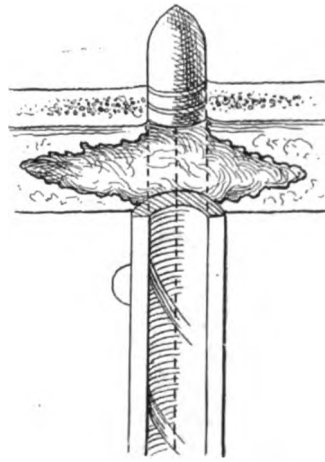


FIG. 3.

PLATE II.



*A*

Wound of entrance in upper chest.



*B*

Smudge mark on hand.

PLATE II.—*A*, Excised wound of entrance in upper chest; preserved in formaldehyde. Close range,—less than 3 inches away,—the flame having singed the lace over the wound; only the larger grains of powder have penetrated into the skin (see Plate I, Fig. 2, *b*). Hole slightly funnel shaped. Edges changed by powder grains and smudged, and with fouling from bullet; also shows elongation due to the surface tension of skin which so often disturbs a wound and which must be overcome before a true estimate of the size and shape of the wound can be obtained. When overcome this was a round hole, the same size as the bullet, into which projected a fringe of pulpified tissue that had before filled the space where the hole now is. *B*, The smudge mark on the left hand made while the hand firmly grasped the revolver about the chamber and barrel, the right hand having been used to grasp the hand-hold and trigger. (Both are from the same case which was one of suicide. *A* is a trifle larger than life; *B* is much reduced.)



PLATE III.—Library card that was 12 inches from muzzle of 0.32 calibre revolver; exact size. The actual hole appears to be 0.175 in. x 0.115 in. The mark shows by accurate measure that a bullet measuring about 0.31 + in. in all diameters passed through and wiped off fouling. The size of the hole accurately measured is 0.31 + in. The photograph was taken so as to intensify the contrasts, by Mr. Charles Pancoast, a most expert photographer, to whose skill I am indebted for all my plates. The powder grains in several places had passed through the card. Two were exploding during their passage through and quite a number exploded against the card. The after-coming gases and powder debris form a mark above and to the left of the hole showing the effect of flip upward and to the left. The flame itself did not reach the card. The card was not large enough to catch all the powder. The increased contrast brings out remarkably the nature of the smudge, showing it to be due to a vast number of fine particles of debris which to the unaided eye show only as a yellowish-gray pastel effect (this Plate to be studied with Plate V).

**PLATE III.**

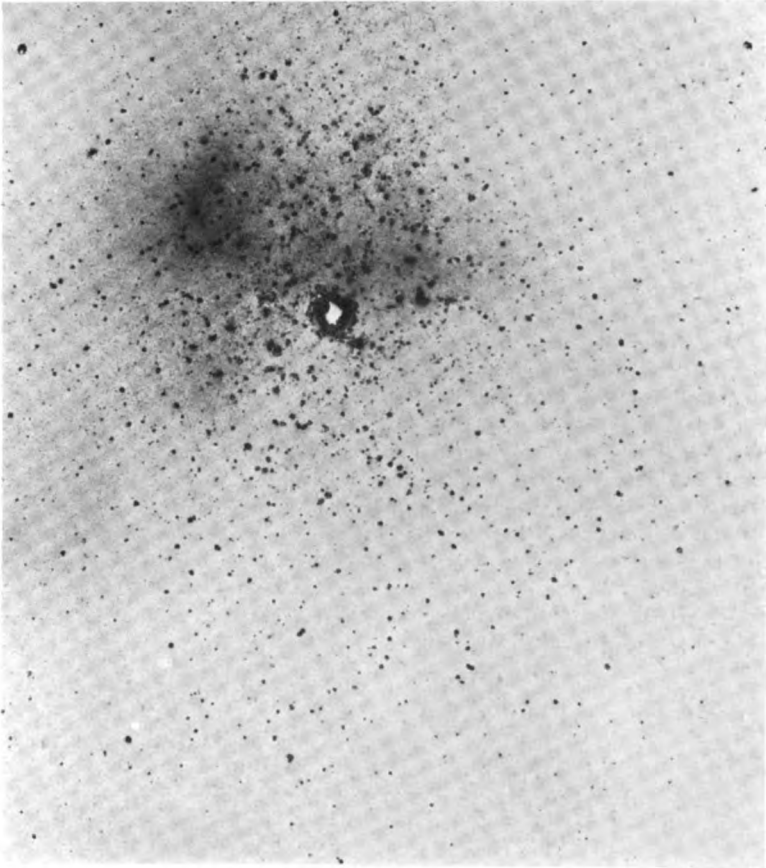


PLATE IV.

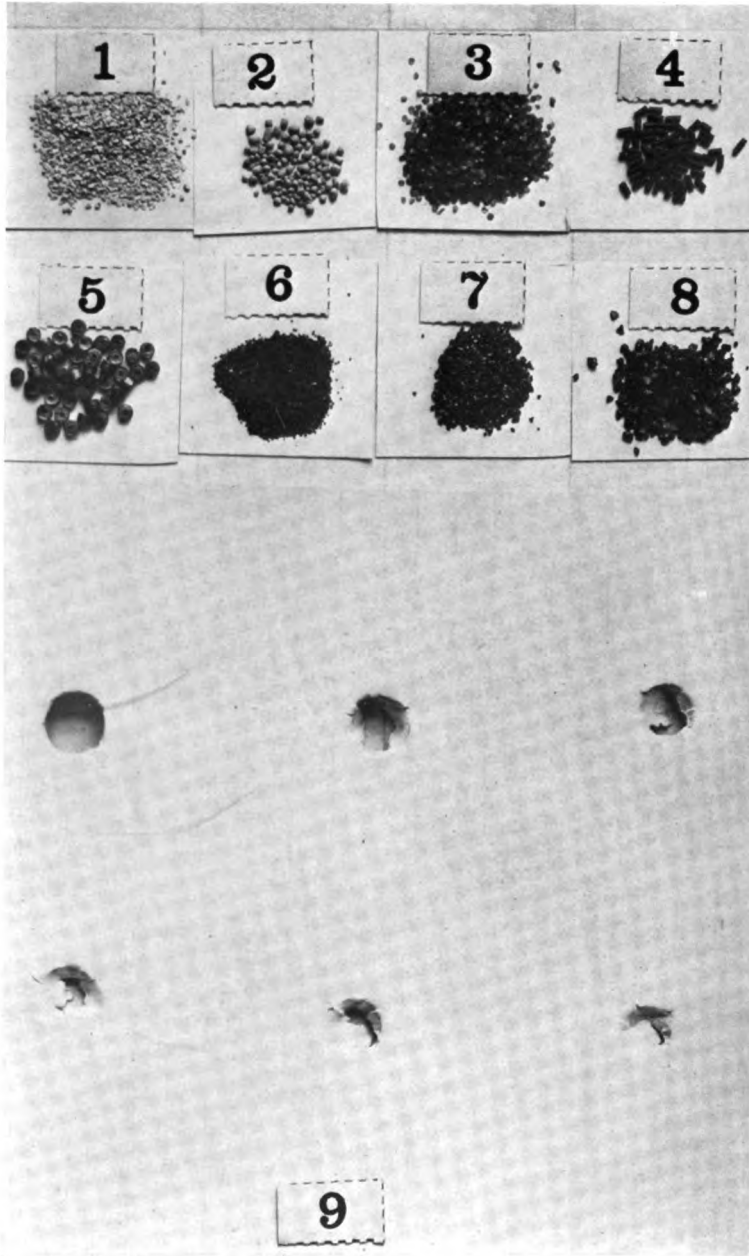


PLATE IV.—Fig. 1.—Smokeless powder; small white granules (see Plate IV, Fig. 3). Fig. 2.—Smokeless powder; medium granules, yellow; from Government blank cartridge. Fig. 3.—Smokeless powder; small flakes, gray; from 0.38 Colt, F. A. (Plate VI, Fig. 20). Fig. 4.—Smokeless powder; small tubes; red; from Springfield 0.308 F. A. (Plate VI, Fig. 17). Fig. 5.—Smokeless powder; beads; black; from "Krag," F. A. (Plate VI, Fig. 16). Fig. 6.—Black powder; very fine grain (French) (Plate VI, Fig. 7). Fig. 7.—Black powder; medium fine grain (German) burnished (Plate VI, Fig. 5). Fig. 8.—Black powder; medium grain (American). Fig. 9.—Six holes made by conical bullet (see Plate VI, Fig. 17), showing types of closure that delude careless persons in measuring. They are all the same size and fit a paper cone the size of the bullet; they illustrate the slit appearances seen in wounds and also the way a filled wound appears smaller. They are made in a library card of fair quality and illustrate conditions commonly found in wounds in the skin (compare with Plate III).

**PLATE V.**—(*Ignited Powder Charges—Exact Size and Same Contrast Intensification as Plate III. Showing the difference between different powders. In each case the charge was put in a small heap and ignited by a burning match.*) Fig. 1 (see Plate VI, Fig. 3 and Plate IV, Fig. 1).—Fine granules of smokeless; full charge; burns very freely and leaves brown residue. Fig. 3 (see Plate VI, Fig. 16, and Plate IV, Fig. 5).—Beads of smokeless; full charge; shows brown scorch and little residue. Fig. 5 (see Plate VI, Fig. 20, and Plate IV, Fig. 3).—Fine scales of smokeless; full charge burned; shows brown scorch and black residue. Fig. 6 (see Plate VI, Fig. 7, and Plate IV, Fig. 6).—Fine grains of black powder; full charge exploded; shows gray streaks and pastel effect, and few burns. Fig. 7 (see Plate VI, Fig. 5, and Plate IV, Fig. 7).—Medium fine black powder; full charge exploded; shows pastel effect and many burnings. Fig. 8 (see Plate IV, Fig. 8).—Medium black powder; 0.38 S. & W.; W. R. A. full charge; shows pastel effect and path of big grains. Fig. 9.—From Peters 0.38 S. & W.; semi-smokeless; shows pastel of black powder and burning of smokeless, both less marked than in single type.

PLATE V.

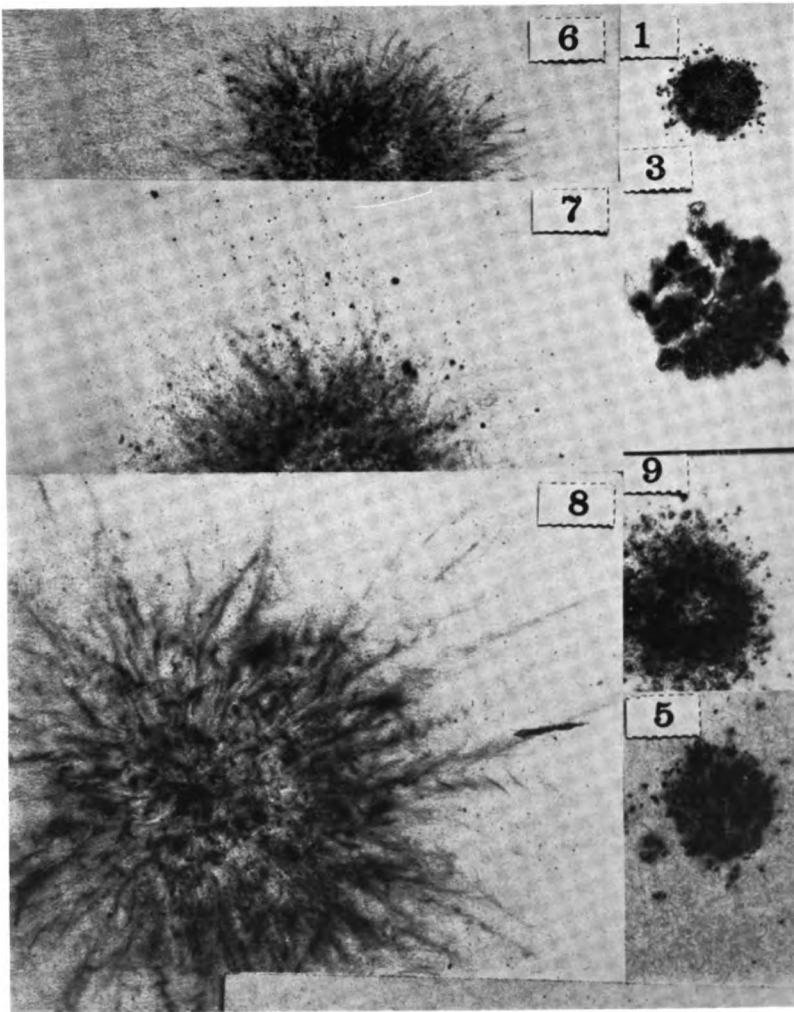


PLATE VI

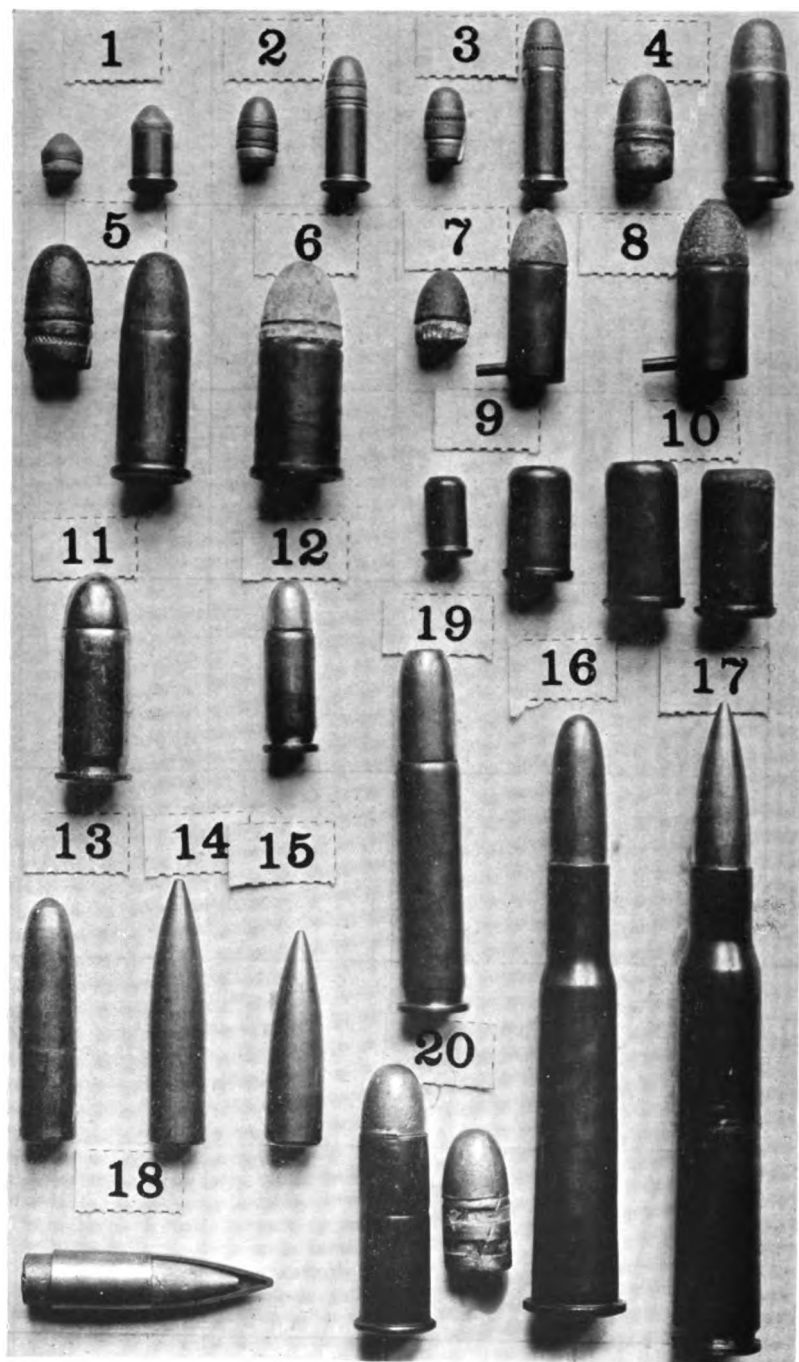


PLATE VI.—*Cartridges and Bullets—Exact Size.*) Fig. 1.—BB cap, W. R. A., 0.22 rim-fire; also bullet from same; weight 19 grains; shows crimp ring. Fig. 2.—0.22 short, W. R. A., rim-fire; also bullet from same; weight 30 grains; shows flat base, crimp ring, milled ring, extra ring, and taper notch. Fig. 3.—0.22 long, W. R. A., rim-fire; also bullet from same; shows deeper seating below crimp ring, milled ring, and taper notch; weight 34 grains. Fig. 4.—0.32 S. & W., U. M. C., centre-fire; also bullet from same; weight 87 grains; 0.313 calibre; shows shallow cup in base, fine milling in lubricant groove, deep seated, crimp groove and taper notch. Fig. 5.—0.38 S. & W., R. W. S. (German), centre-fire, poor work and material; also bullet from same; weight 140 grains; 0.357 calibre; shows slanted milling in lubricant groove; shallow seating. Fig. 6.—0.44 Webley (English type), U. M. C.; shows corrosion (old sample) and old shape. Fig. 7.—BB 7 (French) pin-fire; also bullet from same; weight 46 grains; 0.297 calibre; shows deep base cup, shallow seating, milling, deep crimp groove; corrosion within shell. Fig. 8.—BB 9 (French), pin-fire; a thin copper shell. Fig. 9.—0.22 blank, U. S.; copper shell; rim-fire; shows rough crimp which is much more marked than in ball cartridge. 0.32 S. & W., U. M. C., centre-fire. Fig. 10.—0.38 S. & W., U. M. C.; and after explosion, showing the heavy crimp practically unchanged except for corrosion by products of burning powder. Fig. 11.—F. N., German automatic, jacketed bullet, 0.358 calibre; shows large ejector groove. Fig. 12.—0.25 A. P. C., U. M. C., automatic; jacketed bullet. Fig. 13.—0.308 "Krag" bullet with jacket; weight 220 grains; shows depth of seating; compare with Fig. 16. Fig. 14.—0.308 "Krag" bullet, sharp pointed; weight 202 grains; introduced by private makers; later adopted by U. S. A. Fig. 15.—0.308 Springfield; early pattern of sharp-pointed jacketed bullet used by U. S. A. (see Fig. 17); weight 150 grains. Fig. 16.—"Krag" cartridge, F. A. 03; shows large powder space. Fig. 17.—Springfield cartridge; now used by U. S. A., F. A. 08. Fig. 18.—Dissection to show relation between lead core and composition jacket (compare Figs. 15 and 17). Fig. 19.—0.32 S. H. R., U. M. C.; automatic rifle; jacketed bullet. Fig. 20.—0.38, F. A., 11.03; regulation army Colt revolver; also bullet from same; weight 150 grains; 0.355 calibre; shows deep seating double smooth groove for lubricant.

PLATE VII.—(*Bullets that Have Been Shot—Exact Size.*) Fig. 21.—0.32 calibre; weight 88 grains; wound in temporal bone; marks on nose of bullet from temporal bone; marks on upper side from inside of skull; rebounded into brain substance over 2 inches without causing fracture. Figs. 22 and 23.—Same case; both bullets from same lot of cartridges. Fig. 22.—0.345 calibre (0.38), 142 grains; passed through crest of ilium, great veins in pelvis and intestines; shows cloth marks on nose; marks from rifling of barrel; traces of bone scratches. Fig. 23.—0.34 + (0.38), 140 grains; crushed third and fourth ribs, front of chest; evidently tipped over on third rib, smashing base on fourth; did not penetrate pleura. Figs. 24, 25, 26.—All from same case; same cartridges (0.38). Fig. 24.—Struck upper arm at angle; flattened against humerus; 135 grains, showing loss of weight; shows shred of cloth on nose. Fig. 25.—Passed through heart; 145 grains; shows base altered by so-called soft tissues. Fig. 26.—Entered upper arm; passed through muscle and out; entered chest; shows dent on nose from upper surface of rib and evidence of dirty barrel. Fig. 27.—80 grains, 0.32; close range wound of head; the nose shows smash received by contact with temporal bone; many small fragments of bone were driven into brain substance; a broad ragged path through brain; passing side on and striking inside of skull, rebounding a short distance. Figs. 28, 29, 30, 31.—All from same case; same cartridges; all 0.38, weighing before firing about 144 grains. Fig. 28.—Smashed ribs going in and out of chest, weight 143 grains; shows hollowing of side by scraping by bone; slight marks



on nose. Fig. 29.—Passed through lungs and great vessels shows nose smashed by ribs; milling in lubricant groove; weight 141 grains. Fig. 30.—Entered tip of shoulder; out 1 inch from entrance; into neck, smashing vertebra; shows typical disorganization by vertebra. Fig. 31.—Fragment of tip (nose), weighing 47 grains; bullet having split up on striking femur. Figs. 32, 33, 34.—All from same case; 0.32 calibre, 84 grains; same sort of cartridges; all show deep base cup; milling groove and evidence of dirty barrel, but not choked. (Negro servant shot whole family; two other bullets not shown.) Fig. 32.—Passed through lung. Fig. 33.—Shortened by striking squarely on sternum. Fig. 34.—Passed through heart; evidently turned by soft tissues, striking base against rib at back of thorax. Fig. 35.—0.38 calibre, 128 grains (considerable loss of weight); passed in same direction as it now points (up and to the left of plate), striking the frontal bone, which was very thick and dense, smashing the nose in a way that shows the bone gave as the bullet gave, which caused a curved rather than a flat distortion; the bullet made a complete half turn striking the brain case with its opposite corner, forming a mould of bone (upper right of picture), rebounding into the brain without a second fracture of the skull; an unusually interesting specimen. Fig. 36.—0.32 calibre, 82 grains. Very difficult to follow course; entered back of neck; gas pocket under skin; traced downwards to spine, then lost owing to gas wound; finally found in front of brain and traced back, it having taken three distinct turns, entering the head through the foramen magnum; the mark corresponded with one on the posterior edge of the foramen magnum. Figs. 37, 38.—Same case; both 0.32; same cartridges. Fig. 37 shows rifling marks (compare with Figs. 22 and 29). Fig. 38.—Passed through upper lid of left eye, through orbit to sphenoid bone, under optic chiasm, causing concussion of the brain and hemorrhage from the small branches of the cerebral vessels without itself entering the brain case; shows typical smashing from fine grained hard bone. Fig. 39.—0.32 calibre; caused chest wound; nose marked by rib (compare with Fig. 26); angle of incidence shown in both. Fig. 40.—A typical smash from hard bone that yields at about the same rate as the bullet; make unrecognizable, probably 0.38, weight 92 grains. Figs. 41 and 42.—Same case; 0.32 calibre, 82 grains; same cartridges. Fig. 41.—Nose smashed by second rib in front and tumbling so that sixth rib in back marks base (compare and contrast Fig. 23). Fig. 42 shows dirty barrel with choking, resulting in obliteration of crimp groove and drawing of head into lubricant groove (contrast with Figs. 32 and 33 where barrel was dirty but not choked). Fig. 43.—Base and three fragments; bullet deflected and shaved by edge of orbit; passed through orbit, destroying eye, through floor of anterior fossa, being again deflected, struck the base of the brain case and rebounded at an angle of 45° with brain; the young colored man lived two weeks, finally dying of sepsis. Fig. 44 shows cloth marks on nose due to cloth of coat; very fine example. Fig. 45.—Rear view of 0.38 Colt bullet (see Plate VI, Fig. 20); obtained at range, bullet having struck iron plate square on. Fig. 46 shows two sharp-pointed bullets (compare Plate VI, Figs. 15, 17, 18), smashed by striking hard objects; showing different action on lead and jacket. Figs. 47, 48, 49, 50.—“Krag” bullets (see Plate VI, Figs. 13, 16). Fig. 47.—Shot into soft wood; shows marks of rifling of barrel. Fig. 48 shows bending of spent bullet striking resistant object at an angle. Fig. 49.—Mushrooming, lead all up at nose and jacket burst open. Fig. 50.—Typical explosion of jacket with lead entirely out and fragments of jacket detached.

PLATE VII.





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